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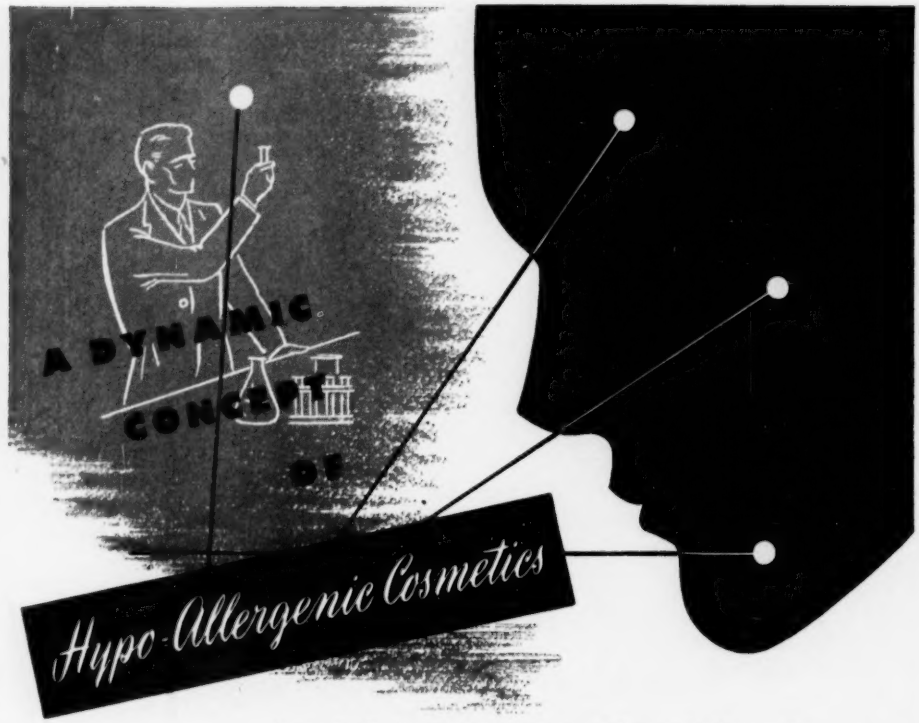

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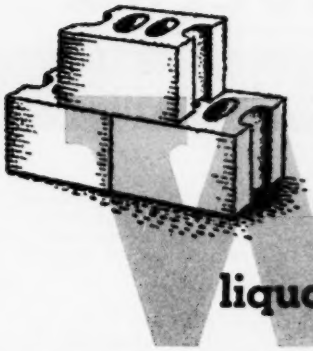

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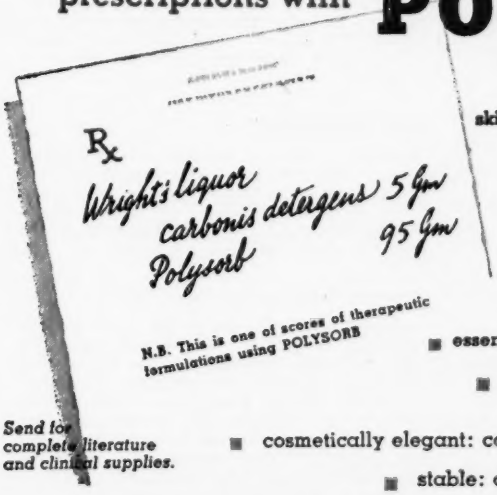
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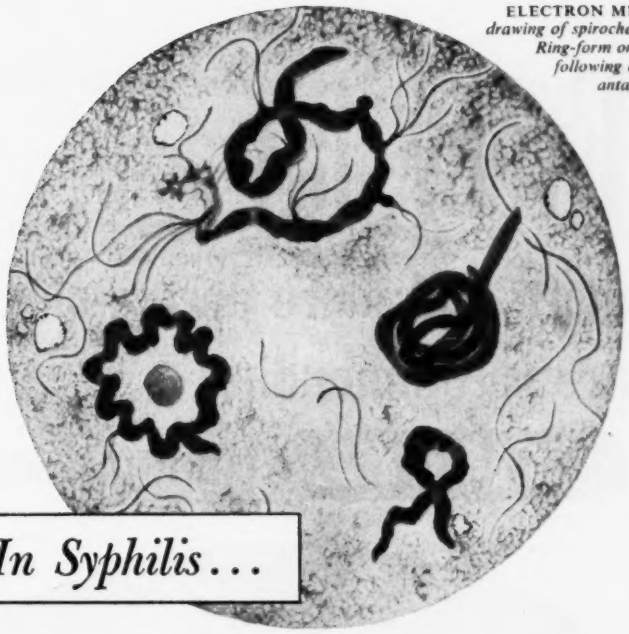

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THE ADVERSE INFLUENCE OF SYPHILITIC INFECTION ON THE LONGEVITY OF MICE AND MEN

PAUL D. ROSAHN, M.D.
NEW BRITAIN, CONN.

SYPHILIS is a chronic granulomatous infection. In man it has the potentiality of producing anatomical alterations in almost all organ systems. It may evoke a variety of clinical symptoms and signs which place it among the easiest, yet sometimes among the most difficult, of diseases to diagnose. Many acquire syphilis, but good evidence [1] indicates that the large majority of syphilitic persons, even if treatment has been denied them, do not die as a direct result of the disease. This report is concerned with mortality among syphilitic subjects and with the influence of syphilitic infection on longevity. The evidence bearing on this problem is based on clinical data and vital statistics, on autopsy observations, and on observations on the induced infection in a laboratory animal, the mouse. This evidence will be presented below.

EVIDENCE FROM CLINICAL FINDINGS AND VITAL STATISTICS

An illuminating group of reports has appeared under the auspices of the United States Public Health Service. The first of these, by Usilton and Miner,[2] described the course of death from all causes, not from syphilis alone, in the white and Negro male population of the United States with acquired syphilis. The data were based on patients with syphilis under treatment or observation for six months or more in five syphilis clinics. Life tables were constructed giving the complete expectation of life for various ages, in comparison with similar values derived from the general population. As shown in Table 1, these authors found a decrease in expectation of life for white men with syphilis varying from 7.0 years at age 30 to 1.5 years at age 60, with an average decrease of 4.8 years. Negro men with syphilis had an average decrease of life expectancy of 7.1 years. As the authors are careful to

Awarded First Prize in the Second Annual Essay Contest of the American Dermatological Association, 1952.

This study was aided by grants from the Research Grants and Fellowships Division, National Institutes of Health.

Read at the Seventy-Second Annual Meeting of the American Dermatological Association, Colorado Springs, Colo., April 23 to 26, 1952.

From the Laboratories of the New Britain General Hospital and the Department of Pathology, Yale University School of Medicine, New Haven, Conn.

1. (*a*) Bruusgaard, E.: Über das Schicksal der nicht spezifisch behandelten Luetiker, Arch. Dermat. u. Syph. **157**:309, 1929. (*b*) Rosahn, P. D.: Studies in Syphilis: VII. The End Results of Untreated Syphilis, J. Ven. Dis. Inform. **27**:293, 1946; (*c*) Autopsy Studies in Syphilis, ibid. Supp. 21, 1947.

2. Usilton, L. J., and Miner, J. R.: A Tentative Death Curve for Acquired Syphilis in White and Colored Males in the United States, J. Ven. Dis. Inform. **18**:231, 1937.

emphasize, this decreased life expectation for syphilitic men was the result of deaths not only directly from syphilis but from all causes. No attempt was made to segregate the deaths which were caused by syphilitic infection per se from those resulting from other disease processes, but the total effect was a profound reduction of life expectancy in the syphilitic population. It is of more than passing interest that the base group of syphilitic patients utilized by Usilton and Miner [2] in constructing their tables was a group under treatment or observation for six months or more in the prepenicillin era.

A series of reports by members of the United States Public Health Service deals with studies of untreated acquired syphilis in the male Negro.[3] The first of these, based on material collected in Alabama from among 1,782 male Negroes aged 25 or more, included 399 syphilitics who had never received treatment and 201 presumably nonsyphilitic Negro men of comparable age. Each man was subjected to a careful clinical survey which included a history, physical examination,

TABLE 1.—*Life Expectancy of the General Population and of the Syphilitic Population**

	White Men				Negro Men			
	Complete Expectation of Life, Yr.		Decrease Among Syphilities		Complete Expectation of Life, Yr.		Decrease Among Syphilities	
Yr. of Age	General Population	Syphilitic Population	Yr.	%	General Population	Syphilitic Population	Yr.	%
30	37.5	30.6	7.0	18.6	30.7	22.9	7.8	25.4
35	33.3	27.0	6.4	19.2	27.5	19.1	8.4	30.5
40	29.2	23.6	5.7	19.5	24.6	16.9	7.7	31.3
45	25.3	20.6	4.7	18.6	21.6	15.6	6.0	27.8
50	21.5	17.9	3.6	16.7	18.8	13.0	5.8	30.9
55	18.0	15.4	2.6	14.4	16.3	10.2	6.1	37.4
60	14.7	13.2	1.5	10.2	13.7	8.2	5.5	40.1
	Average decrease		4.8	17.3	Average decrease		7.1	30.5

* Adapted from Table 4 of Usilton and Miner.[2]

roentgenologic study, spinal fluid examination, and serologic evaluation. In the group of syphilitic patients there were 46.6% with evidence of disease of the cardiovascular system, as compared with 23.9% in the control group. Only 15.8% of the syphilitic group had no evidence of disease in any of several organ systems evaluated, while 61.2% of the nonsyphilitic group had no indication of diseases of these same systems. In those under 40 years of age, only one-fourth with untreated syphilis had no manifestations of disease, whereas three-fourths of the uninfected persons of comparable age gave no evidence of disease. This study focuses attention on the frequency of disease manifestations in the syphilitic host. It is not concerned with mortality but with morbidity. On the basis of these findings, the conclusion is warranted that morbidity in the untreated male syphilitic Negroes far exceeded that in a comparable presumably nonsyphilitic group.

A later report [4] extended these studies to a consideration of the effect of syphilis on the lifespan of the human host. The material was expanded to include the

3. Vonderlehr, R. A.; Clark, T.; Wenger, O. C., and Heller, J. R., Jr.: Untreated Syphilis in the Male Negro: A Comparative Study of Treated and Untreated Cases, J. Ven. Dis. Inform. **17**:260, 1936.

4. Heller, J. R., Jr., and Bruyere, P. T.: Untreated Syphilis in the Male Negro: II. Mortality During 12 Years of Observation, J. Ven. Dis. Inform. **27**:34, 1946.

records of 410 Negro men with untreated syphilis for comparison with the originally described control group of 201 uninfected Negro men. From the time that the study population was originally selected during the winter seasons of 1931-1932 and 1932-1933, through the end of 1944, of the original group of 410 infected men 101, or 24.6%, had died, in comparison with 28, or 13.9%, of the 201 in the uninfected control group. The difference in the mortality rates of the two groups is significant (chi-square = 9.26, $P < 0.01$). In order to measure the relative mortality more exactly, life tables were constructed. The results, summated in Table 2, indicated that syphilitic Negro men aged 25 to 29 years suffered an average loss of eight years of life in comparison with nonsyphilitic persons of the same age, sex, and race. This is a reduction of about 20%. All age groups of syphilitics had a reduced longevity when contrasted with the expectancy of life of the control group. The findings demonstrate that the untreated Negro men of age 25 to 50 years with syphilis had their life expectancy reduced on an average of about 20%.

TABLE 2.—*Life Expectancy of Syphilitic and Nonsyphilitic Negro Men**

Age Period	Average No. of Years of Life Through Age 74 Remaining to Men Alive at Beginning of Age Period		Decrease Among Syphilitics	
	Controls	Syphilitics	Yr.	%
25-29	42.37	34.09	8.28	19.5
30-34	35.58	30.83	7.75	21.8
35-39	34.34	28.18	6.16	17.9
40-44	29.88	24.12	5.76	19.3
45-49	25.37	20.13	5.24	20.7
50-54	20.86	17.28	3.58	17.2
55-59	16.42	14.28	2.14	13.0
60-64	12.17	10.63	1.54	12.7
65-69	8.18	7.17	1.01	12.3
70-74	4.33	4.06	0.27	6.2
Average decrease			4.17	16.1

* Adapted from Table 1 of Heller and P. T. Bruyere.[4]

The next study in the series, by Deibert and M. C. Bruyere,[5] carried the investigation still further into the area of morbidity. In 1938 and 1939, a second physical examination was made on as many of the original group as could be reached. Of these, 155 were untreated syphilitics, 115 were infected but had received some treatment, and 153 were believed to be nonsyphilitic. In this reexamination special emphasis was directed to the cardiovascular system. Examination of the radial, brachial, and temporal vessels revealed evidence of arteriosclerosis more frequently among the syphilitic patients than among the nonsyphilitic control group. For example, in the age group 35 to 64 years, examination of the temporal vessels showed arteriosclerosis in 56 of 108 syphilitics, or 51.9%, whereas only 30, or 27.8%, of the 108 nonsyphilitic controls had changes in these arteries (chi-square = 13.06, $P < 0.01$). This is a significant difference. Abnormal conditions of

5. Deibert, A. V., and Bruyere, M. C.: Untreated Syphilis in the Male Negro: III. Evidence of Cardiovascular Abnormalities and Other Forms of Morbidity, J. Ven. Dis. Inform. **27**:301, 1946.

the lymph nodes were significantly more frequent in the syphilitic than in the control group. Syphilitics exhibited more loss of vision at all ages than did the controls. Significant differences could not be demonstrated as regards genitourinary diseases, but in every age group the small differences observed were in the direction of a higher frequency of genitourinary abnormalities in the syphilitic group than in the syphilis-free group. It was concluded from this study that an untreated syphilitic population exhibits a greater frequency of morbid states than an uninfected population of similar characteristics and environment.

The most recent report [6] in this series was published in 1950. This study was an attempt to estimate the increment of abnormalities among the untreated syphilitics and nonsyphilitic controls occurring during the 16-year period following the original examinations. In the fall of 1948 a third physical examination was performed on all the original group who could be located, a total of 155 men. When the effect of differences in age distribution between the untreated syphilitics and the nonsyphilitic controls was removed by a standardization procedure, significant differences in the combined mortality and morbidity between the two groups were demonstrated.

All the last four reports by the Public Health Service group were based on an untreated population of syphilitic Negroes. Another study by Smith and M. C. Bruyere [7] compared the mortality in a general population with the mortality in that portion of the same population which was known to be infected with syphilis and which had received active treatment in the prepenicillin period. The syphilitic group was drawn from the records of the Department of Dermatology and Syphilology of the University of Virginia Hospital, Charlottesville, and the general population from the surrounding area of 11 counties. From 1920 through 1941 the University Hospital admitted 2,908 patients with acquired syphilis who were 15 years of age or older. The average period of observation of these patients was about four years. Comparison of the general with the syphilitic populations was made by means of the temporary expectation of life at various ages obtained from abridged life tables. These were prepared for white and for Negro persons of both sexes. In each of the four race-sex groups under consideration, the life expectancy of the syphilitic population was lower at every age period than that of the general population. For white men in the age group 25 to 29 years, the temporary expectation of life for the syphilitic was 29.16 years, as contrasted with 36.54 years for the general population. This constitutes a reduction in the life expectancy of the syphilitic group of about seven years, or 20%, a finding identical with that of Heller and P. T. Bruyere.[4] The data indicate that the average lifespan of persons with syphilis under routine treatment was shorter than that of uninfected persons.

The observations of Martin [8] add further evidence for the conclusion that syphilis is associated with an unfavorable influence on longevity. This investigator

6. Pesare, P. J.; Bauer, T. J., and Gleeson, G. A.: Untreated Syphilis in the Male Negro: Observation of Abnormalities over 16 Years, Am. J. Syph. **34**:201, 1950.

7. Smith, D. C., and Bruyere, M. C.: The Effect of Treated Acquired Syphilis on Life Expectancy, J. Ven. Dis. Inform. **27**:39, 1946.

8. Martin, H. W.: The Effect of Syphilis on Nonwhite Longevity as Reflected in Mortality Data, Georgia, 1939-41, J. Ven. Dis. Inform. **32**:57, 1951.

studied the vital statistics of the State of Georgia to determine to what extent syphilis as a reported cause of death curtailed life expectancy among nonwhites during 1939 to 1941. Two types of life tables were constructed. The first was a "regular" life table based on mortality rates from all causes, and the second was a "special" life table in which syphilis as a reported cause of death was eliminated. Such tables for nonwhite men and women revealed that in every age through the age group 85 to 89, all values of the special life table were consistently higher than those of the regular table. The differences are considered to be the result of syphilis as a reported cause of death. For example, the average future lifetime of the nonwhite male at birth was shortened by 0.82 year because of syphilis. These findings, it should be noted, relate to syphilis as a reported cause of death. As a result, they exclude all morbid processes other than those of a syphilitic nature which may occur in syphilitic persons and which may affect their mortality rates.

The pattern of all the evidence cited in this section clearly indicates that mortality among syphilitics is higher than among nonsyphilitics and, as a corollary, that the longevity of persons harboring syphilitic infection is reduced in comparison with that of syphilis-free persons. This generalization is applicable to the four sex-race groups under discussion, and apparently in the prepenicillin era was not altered by the treatment or lack-of-treatment status of the syphilitic patient. Evidence is not available by which it can be determined whether the reduced longevity of untreated syphilitic patients was the result of absence of therapy, or whether the reduced longevity of treated patients was related to exposure to treatment itself. Unfortunately, none of the studies were planned to investigate these aspects of the problem. In order to answer them, it would be desirable to study three parallel groups, namely, untreated syphilitic patients, treated syphilitic patients, and nonsyphilitic persons, and this information is not presented in any of the cited reports.

Finally, these studies tend to indicate that morbid processes other than those directly attributable to syphilitic infection are more frequent among syphilitics than among those free of the disease. These morbid processes, alone or in combination with syphilitic infection, are perhaps responsible for the reduced longevity of syphilitic patients. Does syphilis, a specific infection, produce a nonspecific depression of host resistance to other disease processes, and is this hypothetical inhibition of immunity, in association with the syphilitic infection itself, an important factor in reducing the longevity of the syphilitic person? If syphilitics do have an increased susceptibility to other disease processes, is this the result of the infection itself, or rather does it stem from the environmental factors which favor the acquisition of syphilis? The evidence from autopsy material may throw some light on these aspects of the problem.

EVIDENCE FROM AUTOPSY FINDINGS

In a previous report [9] it was demonstrated that fewer persons with morphologic evidence of syphilis at autopsy survive the age of 70 than do nonsyphilitics in the same autopsy populations. This report summarized the data presented by Teodori,[10]

9. Rosahn, P. D., and Black-Schaffer, B.: (*a*) Studies in Syphilis: I. Review of Incidence of Syphilis in Autopsies on Adults, Arch. Int. Med. **72**:78, 1943; (*b*) III. Mortality and Morbidity Findings in the Yale Autopsy Series, Yale J. Biol. & Med. **15**:587, 1943.

10. Teodori, U.: Rilievi statistici concernenti la sifilide nel materiale autoptico del' Instituto di Anatomia Patologica di Firenze dal 1918 al 1935 con rilievi comparativi sulla frequenza dei tumori maligni e della tubercolosi, Dermosifilografo **13**:143, 1938.

Bell,[11] Frates,[12] Melchior,[13] and Koppisch.[14] These findings, in somewhat different form, are given in Table 3. Of a total population of 40,729 persons, 1,692, or 4.15%, had lesions of syphilis which were identified at autopsy. In the group without syphilitic lesions, there were 18.6% who had survived the age of 70. In the group with syphilitic lesions, the significantly lower proportion of 7.2% had lived to the age of 70 or over. Even though the authors utilized varying criteria for the morphologic diagnosis of syphilis, the conclusion appears justifiable that fewer persons with anatomical lesions of syphilis lived to the age of 70 or over than did persons in the same autopsy series in whom syphilitic lesions could not be identified.

A critical evaluation of this conclusion suggests the possibility that it is the result of a bias in the constitution of the autopsy populations. If persons, upon the diagnosis of syphilis, were transferred to other hospitals and thus were removed from the populations providing the autopsy material or if, conversely, the autopsy populations were drawn from hospitals devoted especially to geriatric problems, it is conceivable that a spurious difference in the age constitutions of the syphilitic and nonsyphilitic groups might result. Internal evidence in the basic reports

TABLE 3.—*Age Distribution of Persons With and Without Syphilitic Lesions at Autopsy**

	Age, Yr.					
	20-69.9		70 and Over		Total	
Group	No.	%	No.	%	No.	%
Nonsyphilitic	31,772	81.4	7,265	18.6	39,037	100.0
Syphilitic	1,570	92.8	122	7.2	1,692	100.0

* Adapted from Table 4 of Rosahn and Black-Schaffer.[9a] Data are summated from the reports of Teodori,[10] Bell,[11] Frates,[12] Melchior,[13] and Koppisch.[14] Data for women published by the last-named author have not been included because of the small number of women in his series.

suggests that this sampling error was not operative in the selection of the material. If such bias did exist, it was present to the same extent in the Yale autopsy series, and this, it is believed, was not the case.

The studies of syphilitic infection based on the Yale autopsy series have been described in earlier reports.[15] In brief, there were 3,907 necropsies on persons aged 20 years and over in the period Sept. 22, 1917, to March 8, 1941. From these were selected all cases, 380 in number, with the diagnosis of syphilis on clinical or laboratory or postmortem evidence. Two control groups, *A* and *B*, were selected from the nonsyphilitic residue, having the same temporal distribution in the autopsy series as the syphilitic group. Since it was demonstrated that Negroes, both syphilitic and nonsyphilitic, died at a significantly earlier mean age than whites, they were

11. Bell, E. T.: Frequency with Which Syphilitic Lesions Are Encountered in Postmortem Examinations, Arch. Path. **26**:839, 1938.

12. Frates, A.: Considerazioni statistiche sulla sifilide nelle prime 10,000 autopsie dell' Instituto di Anatomia Pathologica della r. Universita di Milano, Clin. med. ital. **65**:1015, 1934.

13. Melchior, L.: Om sektionsfund og dødsaarsager hos patienter med erhvervet syfilis, Ugesk. læger **84**:1351, 1922.

14. Koppisch, E.: La sifilis en Puerto Rico: Estudio basado en la revision de 1,000 autopsias consecutivas (informe preliminae), Bol. Asoc. méd. Puerto Rico **31**:160, 1939.

15. Rosahn.[1c] Rosahn and Black-Schaffer.[9]

not included in the following tabulations which are presented in slightly different form from that of the tables originally published.

Table 4 summarizes the age distribution of the syphilitic group with lesions at autopsy, in comparison with the age distribution of the nonsyphilitic Control Groups *A* and *B*, only whites being considered. It is apparent that fewer syphilitics with anatomical lesions survived the age of 70 than did nonsyphilitics, and the difference has statistical significance. The proportion of nonsyphilitics and of syphilitics with lesions who lived to be 70 or over as observed in the Yale series is surprisingly similar to the proportions summated from five other investigations which are shown in Table 3. This similarity gives added weight to the findings and suggests that if any sampling errors did occur they were equally operative both in the summated series and in the Yale data.

TABLE 4.—*Age Distribution of White Nonsyphilitic Persons and of White Persons with Syphilitic Lesions in Yale Autopsy Series**

Group	Age, Yr.				Total	
	20-69.9		70 and Over			
	No.	%	No.	%	No.	%
Nonsyphilitic						
Control A	285	80.3	70	19.7	355	100.0
Control B	295	83.6	58	16.4	353	100.0
Syphilitic with lesions	108	90.8	11	9.2	119	100.0

* Adapted from Table 8 of Rosahn and Black-Schaffer.[98]

TABLE 5.—*Age Distribution of White Nonsyphilitic Persons and of White Syphilitic Persons Without Syphilitic Lesions in Yale Autopsy Series**

Group	Age, Yr.				Total	
	20-69.9		70 and Over			
	No.	%	No.	%	No.	%
Nonsyphilitic						
Control A	285	80.3	70	19.7	355	100.0
Control B	295	83.6	58	16.4	353	100.0
Syphilitic without lesions	142	90.4	15	9.6	157	100.0

* Adapted from Table 8 of Rosahn and Black-Schaffer.[98]

One other finding of interest has been observed in an analysis of the Yale material. This is shown in Table 5. Of the 380 syphilitic persons in the series, 157 white patients, for whom the diagnosis of syphilis was made on good historical, clinical, or laboratory data, at autopsy failed to show any morphologic tissue changes consistent with a diagnosis of syphilis. Of this group, 9.6% were 70 years of age or over, while among the nonsyphilitic persons comprising Control Groups A and B there were 19.7% and 16.4%, respectively, who had attained or passed the age of 70 before dying. These values for the syphilitic and nonsyphilitic groups are significantly different. A comparison of Tables 4 and 5 shows that, considering the two groups, white syphilitics with syphilitic lesions at autopsy and white syphilitics without morphologic lesions of syphilis at autopsy, the same proportion, about 9%, lived to be 70 years of age or older. In both of these groups, namely,

those with syphilitic lesions at autopsy and syphilitics in whom anatomical evidence of syphilis at autopsy was absent, the proportion living to the age of 70 or over was about half the proportion of the nonsyphilitic group who attained or surpassed the age of 70 before dying. The findings suggest that even though syphilitic lesions do not develop or, having occurred, completely regress before death, the very fact of having been infected reduces longevity. This apparently corroborates the suggestion made in the previous section that syphilis is associated with a nonspecific depression of resistance to other disease processes. However, it does not exclude the possibility that external environmental factors are partly or even wholly responsible for the reduced longevity of the infected group.

Schamberg [16] has drawn attention to a possible bias in the selection of our syphilitic population. He notes that many of the cases were included in the study solely as the result of a positive serologic test for syphilis. Further, he comments, since syphilitics frequently become seronegative either after treatment or spontaneously, it is probable that the original autopsy population included a number of seronegative syphilitics over 70 years of age without lesions, and that these are absent from the autopsied syphilitic group. It is probable, he concludes, that the smaller percentage of syphilitics without lesions in the 70-year group is, at least in part, an artifact caused by spontaneous or treatment-induced seronegativity in the patients in this age group. Although an unknown number of patients of the type described by Schamberg were probably missed, many seronegative syphilitics without autopsy lesions were included in our syphilis group on the basis of a history of a primary lesion, or of treatment, or of a previously positive serologic test. Moreover, many seropositive latent syphilitics were probably excluded, because the serologic status of more than 10% of our basic autopsy population was not known. The magnitude of these factors, the exclusion of both an unknown number of seronegative syphilitics and an unknown number of seropositive latent syphilitics, cannot be estimated; it is entirely likely that they counterbalance each other.

The evidence from clinical studies and vital statistics and from autopsy data presented to this point indicates that syphilitic infection is associated with a reduction in life expectancy. Although differences in the longevity of syphilitic and syphilis-free groups have been demonstrated, these differences need not necessarily be the result of syphilitic disease. They may have been produced by other unknown and unrelated factors. Perhaps the environmental and social conditions which contribute to the probability of acquiring syphilis are such as to have an adverse influence on longevity.

Certain it is that syphilis prevalence rates vary with environment, with education, and with social factors, such as the marital state. On the basis of a survey of the Baltimore Eastern Health District, Clark [17] noted higher rates among Negroes than among whites, and among divorced and separated persons than among married or single ones. The rates also varied directly with educational attainment, the lowest rate occurring among those with college education. An analysis according to type of employment showed the highest rates to be among the emergency employed.

16. Schamberg, I. L.: The Prognosis of Syphilis: A Critical Review of Clinical, Autopsy, and Life Insurance Studies, Am. J. Syph. **29**:529, 1945.

17. Clark, E. G.: Studies on Syphilis in the Eastern Health District of Baltimore City: VI. Prevalence in 1939, by Race, Sex, Age, and Socioeconomic Status, Am. J. Syph. **29**:455, 1945.

Not only do syphilis prevalence rates vary with social and economic factors, but a parallel variation has been noted by Schamberg [16] in the mortality rates from all causes and from seven diseases other than syphilis. His published tables, which are briefly summarized in Table 6, show that among laborers and unskilled workers there are both higher syphilis prevalence rates and higher mortality rates from all causes and from heart disease, pulmonary tuberculosis, cancer, pneumonia, nephritis, suicide, and accident, than among professional and semiprofessional groups. Further, he has gathered statistics which indicate that death rates from syphilis itself are highest among laborers and unskilled workers and lowest among professional groups.

We have now arrived at the very crux of the problem. Syphilis is more frequent among those social, economic, and racial groups in which death rates from all causes are highest. Is the reduced longevity among syphilitic persons, which has been amply demonstrated by clinical and autopsy surveys, the result of syphilitic

TABLE 6.—*Prevalence of Syphilis Among Selective-Service Registrants and Standardized Death Rates, by Occupation**

Occupational Group	Prevalence of Syphilis per 1,000, Age 26 to 30	Occupational Group	Standardized Death Rates per 100,000 Gainfully Occupied Males, 15 to 64 Yr. of Age, All Causes
Professional and semiprofessional.....	16	Professional men.....	670.5
Proprietors, managers, and officials.....	21	Proprietors, managers, and officials...	792.5
Craftsmen, foremen, etc.....	34	Skilled workers and foremen.....	828.9
Service workers.....	47	Semiskilled workers.....	1,009.3
Laborers	52	Unskilled workers.....	1,447.7

* Adapted from Table 4 of Schamberg.[16]

infection, or does it rather stem from the poor hygiene, poor nutrition, faulty sanitation, and the unhealthy conditions in general to which those groups with highest syphilis prevalence rates are probably subjected? The answer is not readily apparent from the evidence now available, and to seek it attention was directed to a study of syphilis in a laboratory animal, the mouse.

EVIDENCE FROM EXPERIMENTAL SYPHILIS OF THE MOUSE

The two previous sections have summarized the evidence from which it is concluded that syphilitic infection of the human host is associated with a decrease in longevity. This evidence in brief is as follows:

1. Expectation-of-life tables for treated syphilitic and nonsyphilitic persons in the general population, for untreated syphilitic and nonsyphilitic Negroes in the same geographic area, for treated syphilitic persons and the general population of another geographic area, and for total population and total population excluding syphilitics in a third geographic area, all reveal a reduction in life expectancy in the syphilitic as compared with the nonsyphilitic groups.

2. Autopsy studies indicate that fewer syphilitics with lesions at autopsy survive the age of 70 than do nonsyphilitics in the same autopsy populations and, further,

that fewer syphilitics in whom lesions are absent at autopsy live to be 70 years of age or over than do nonsyphilitic persons in the same autopsy population.

3. Further, syphilis prevalence rates are highest in the socioeconomic groups in which mortality rates from all causes, and from several major diseases, are highest.

The problem, as stated, is to determine (1) whether syphilis produces a lowered resistance to other mortal diseases, thus accounting for the reduced longevity of syphilitics as a class, or (2) whether the reduced longevity of syphilitics as a class is the result of the increased morbidity and mortality in the socioeconomic groups in which syphilis prevalence rates are highest. The available surveys on the human host are not sufficiently controlled to permit of any precise evaluation of these two alternatives, and for this reason attention was directed to a study of the disease in an experimental animal. The study envisaged the observation of the experimental animal throughout its life to spontaneous death, under controlled laboratory conditions.

The mouse was chosen for the study for two reasons: 1. The lifespan of the mouse, usually less than three years, permits the observations to be pursued within reasonable time limits. 2. The inoculated mouse never develops demonstrable syphilitic lesions, although treponemes can be recovered from the host through tissue passage into rabbits. In this sense syphilis of the mouse is the analogue of true latent syphilitic infection in man. Aside from these considerations, mice with established genetic patterns are available, they produce bountiful numbers of offspring, and their care, feeding, and segregation are easily surmountable problems.

Materials and Methods.—A. Animals: The mouse strain employed throughout stemmed from a group of C57 black mice of Line 6, obtained from the Roscoe B. Jackson Memorial Laboratory in October, 1947. C57 black animals were selected both because of their known genetic pattern and because of the comparatively low incidence of spontaneous tumors in this strain. The experimental animals were derived from the original stock and by subsequent back-cross and brother-sister matings. All animals used in the experiment were virgin; breeding stock was excluded from the investigation.

At the time of weaning, the animals were paired, brother with brother from the same litter, and sister with sister from the same litter. Each animal of a pair was housed in one of two compartments of a cage; the other animal of the pair in the second compartment (Figs. 1 and 2). The cages were cubes constructed of sheet metal and measured 6 in. (15.2 cm.) on a side. Each was provided with a partition dividing it into two compartments of equal volume. Each member of the pair occupied one of these compartments. The cage housing a pair of animals had a mesh screen cover, through the center of which a feeding trough projected into the cage. The feeding trough, which measured 2 by 2 by 3½ in. (5 x 5 x 8.9 cm.), rested on a cutout in the dividing partition in such a way that half of the trough projected into each of the two compartments. By this device food from a common source was available to each compartmentalized member of a pair. The cage construction with the dividing metal partition and the central feeding trough effectively prevented contact between the two members of the pair in each cage. The mice were fed a diet of commercial pellets and received a free supply of water. All animals were maintained in air-conditioned rooms having a temperature thermostatically controlled at 65 to 70 F. Systematic care was observed never to move one animal of a pair without simultaneously and similarly moving the cage containing its mate. This precaution was especially important when cleaning and changing cages and when transporting animals to the room where all inoculations were made. After weaning, the animals were maintained in this fashion throughout their lives, each animal in a compartment of a cage which housed a littermate of the same sex in its second compartment.

The rabbits used for testing the inoculum and for a check on the persistence of the infection in the inoculated mice were all males of miscellaneous breed. They were housed in individual cages and were fed commercial food pellets with a free supply of water.

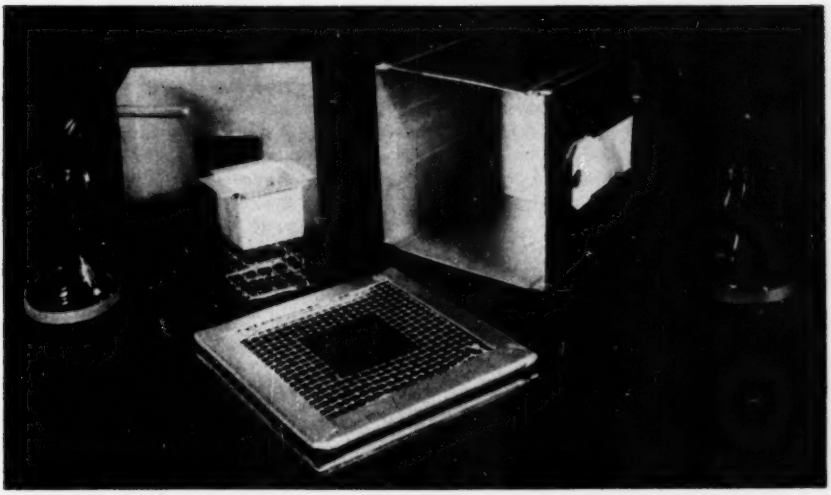

Fig. 1.—The mouse cage disassembled.

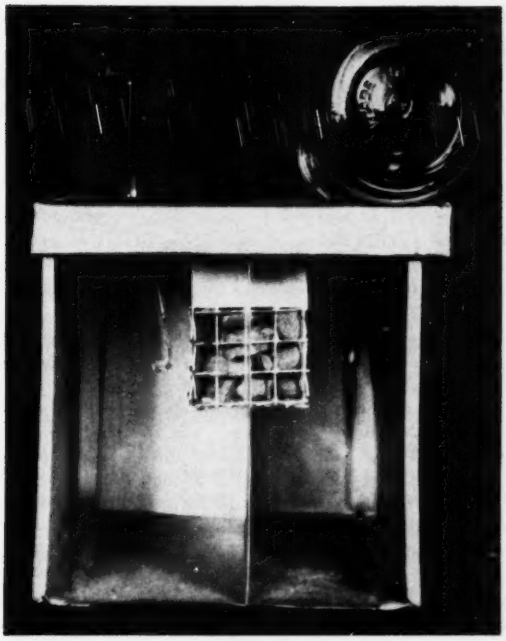

Fig. 2.—Mouse cage, showing the two compartments. One side of the cage and one side of the partition have been removed.

B. Inoculations: As the breeding program provided pairs of animals, they were so grouped for inoculation. Twelve experimental groups were inoculated as shown in Table 7. The age of the animals at the time of inoculation was variable and depended in large measure on the availability of a group of sufficient size to warrant inoculation. Similar procedures were followed in the inoculation of each group.

Rabbits infected with the Nichols strain of the Treponema pallidum served as the source of the infectious agent. Each experimental group of mice received material obtained from a fresh source. This consisted of an active testicular syphiloma from routine passage rabbits. The excised syphiloma was minced and ground in a mortar with saline solution. When the heavier particles had sedimented to the bottom of the mortar, the supernatant saline emulsion was aspirated into a 1-cc. tuberculin syringe equipped with a 24-gauge hypodermic needle, and 0.2 cc. of the emulsion was inoculated intraperitoneally into the experimental animal. After the group of experimental animals had been so inoculated, the control brother or sister of each was similarly inoculated intraperitoneally with 0.2 cc. of saline solution. In subsequent discussions the mice inoculated with treponemes are designated "experimental," and those inoculated with saline solution are called "control." Table 7 shows the different experiments, the dates of inoculation,

TABLE 7.—*Number and Sex of Mice Inoculated, Date of Inoculation, and Dose of Treponemes in Millions per Cubic Centimeter*

Group	Date of Inoculation	Pairs of Males	Pairs of Females	Treponemes, Million/Cc.
A	March 29, 1948	5	2	6.5
B	May 5, 1948	8	9	19.0
C	May 20, 1948	5	10	14.0
D	July 1, 1948	5	5	12.6
E	July 27, 1948	1	4	32.7
F	Dec. 14, 1948	3	4	22.0
G	Sept. 24, 1948	6	5	7.6
H	Oct. 13, 1948	1	3	13.6
I	Dec. 14, 1948	3	3	22.0
J	Jan. 17, 1949	6	6	7.4
K	March 16, 1949	15	18	6.5
L	June 1, 1949	31	25	6.1
	Total	89	94	...

the number of experimental animals by sex in each group, and the treponeme count in millions per cubic centimeter of the different inocula. A total of 89 males and 94 females was inoculated with T. pallidum, each controlled by a littermate of the same sex inoculated with saline solution.

The infectivity of the inoculum employed for each group of experimental mice was determined by inoculating three test mice in addition to those included in the experimental group. These test animals each received 0.2 cc. of the treponeme-laden inoculum intraperitoneally. At periods ranging from 29 to 35 days after inoculation, the test mice were killed by ether inhalation, and all available lymph nodes were removed as described elsewhere.[18] These nodes included the inguinal, periaortic, axillary, and subclavian groups. The nodes from each mouse were pooled in a mortar and ground with 1 cc. of saline solution, and the entire emulsion from a single mouse was inoculated into the right and left testes of a single rabbit. All rabbits so inoculated, a total of 36, exhibited dark-field-positive lesions at the site of inoculation, demonstrating that all the original inocula were infectious.

C. Postinoculation Procedures: Each cage contained two animals, one inoculated with treponemes, the other a littermate of the same sex inoculated with saline solution. Cages were inspected twice a day, and, when an animal was found dead, the date of death was noted. All animals that died were subjected to autopsy examination which included histologic studies when indicated. After the original inoculations, all animals were permitted to live out their lifespans without interruption.

18. Rosahn, P. D.; Gueft, B., and Rowe, C. L.: Experimental Mouse Syphilis: I. Organ Distribution of the Infectious Agent, Am. J. Syph. **32**:327, 1948.

D. Analytical Procedures: At the conclusion of the experiment there were available for analysis a total of 89 male syphilitic mice, each with a control littermate brother, and 94 female syphilitic mice, each with a control littermate sister. In the final tabulations, except where indicated, certain of these pairs were excluded from consideration for the following reasons:

1. Pairs excluded because the experimental mouse was killed for test of infection: As indicated earlier, three mice in addition to those in each experimental group, were inoculated with each of the several treponeme-containing emulsions. Tissues from these mice were subsequently inoculated into rabbits, and the infectiousness of the different inocula was confirmed by the development of dark-field-positive lesions in all these rabbits. These mouse-rabbit transfers were made not later than 35 days after the original mouse inoculations. Although this procedure indicated the infectiousness of the various inocula, it gave no assurance that the inoculated mice in the long-term experiment continued to harbor the infection. It was, therefore, desirable to determine whether syphilitic infection of the experimental mice persisted throughout the life of the animal. To study this point, five aged syphilitic mice whose control nonsyphilitic mates had already died, were killed terminally. The emulsified lymph nodes from each mouse were inoculated intratesticularly into a single rabbit. Each of the rabbits showed dark-field-positive lesions. As shown in Table 8, the infected mice used for this test varied in age from 700 to 919 days, and the period from inoculation to death varied from 642 to 872 days. These five

TABLE 8.—*Syphilitic Mice Killed at Various Periods Following Inoculation to Test Persistence of Infection by Transfer to Rabbits*

Sex	Birth to Death, Days	Inoculation to Death, Days
Male	768	710
Male	717	680
Male	700	642
Male	919	872
Female	703	665

experimental mice, four males and one female, and their controls, were eliminated from the final tabulations, leaving 85 pairs of male mice and 93 pairs of female mice.

2. Pairs excluded because of death within forty days of inoculation: Preliminary scatter diagrams were constructed to indicate the range of life in days following inoculation. This showed a definite concentration of deaths within 40 days after syphilis inoculation. In group K, for example, of 15 male mice inoculated with infectious organisms, 6 died within 40 days of inoculation, and, in the same group, of 18 females inoculated, 9 were dead 40 days later. Obviously a break in technique had occurred in this experiment, and pathogenic organisms other than T. pallidum had probably been injected. Since our purpose was to determine whether an established syphilitic infection had any influence on the longevity of the host, the inclusion of animals dying shortly after inoculation would have introduced a bias in favor of the nonsyphilitic controls. For this reason, all animals, both syphilitic and nonsyphilitic which died within 40 days after intraperitoneal inoculation, were excluded from the final tabulations. Excluded also were the paired mates of these animals, whether syphilitic or nonsyphilitic. There were 11 male syphilitic animals and 3 male nonsyphilitic animals which died within 40 days following inoculation. These 14 mice, together with their paired cage mates were excluded, leaving a residue of 71 pairs of male animals. Similarly, of the female mice, 19 syphilitic and 5 nonsyphilitic animals died within 40 days of inoculation. These 24 mice and their paired cage mates were also excluded from the final tabulations, leaving a balance of 69 pairs of female mice for consideration.

Comparisons were made by recognized statistical methods. Significance was attached to all values of $P \leq 0.01$, that is, where the probability of occurrence through chance alone was 1 or less than 1 in 100.

Results.—A. Age at Time of Inoculation in Relation to Longevity: Each experimental and each control mouse constituting a pair was inoculated on the same day, one with treponemes, the other with saline solution, but the age of different pairs at the time of inoculation varied considerably. The youngest pair of mice when inoculated was 33 days of age, and the oldest pair was 238 days. The mean age of the male mice at the time of inoculation was 79.9±4.1 days, while the female mice had a mean age when inoculated of 92.0±6.6 days. There is no significant difference between these two values. Since the range of ages at inoculation was more than 200 days, which represents about 20% of the age of the oldest mouse at death, it was important to determine whether longevity was influenced in any way by the age the mouse had attained at the time of exposure to infection. In order to answer

TABLE 9.—*Coefficients of Correlations Between Age at Inoculation and Age at Death in Male and Female Syphilitic and Control Mice*

Sex	Inoculum	Group	r	P
Male	Treponemes	Experimental	+0.1911	>0.05
Male	Saline	Control	+0.1528	>0.05
Female	Treponemes	Experimental	−0.0946	>0.05
Female	Saline	Control	+0.1556	>0.05

TABLE 10.—*Mean Length of Life of Male and Female Syphilitic and Nonsyphilitic Mice*

Sex	Male		Female	
Group	Syphilitic	Nonsyphilitic	Syphilitic	Nonsyphilitic
No. of mice	71	71	69	69
Mean in days	502.8	591.7	614.6	694.9
Standard error of mean	20.2	25.4	23.1	22.6
Difference	88.9 ± 32.4 days		80.3 ± 32.3 days	
t	2.74		2.49	
P	<0.01		0.01	

this question, coefficients of correlation were calculated between age at the time of inoculation and age attained at death. These are shown in Table 9. One would expect that the age at death of nonsyphilitic mice would not be influenced by the age of the mouse at the time of intraperitoneal inoculation with saline solution. That this was the case is indicated by the fact that the correlation coefficients for both control groups, males and females, were not significant (males: $r = +0.1528$, $P > 00.5$; females: $r = +0.1556$, $P > 0.05$). In other words, the two variables, age at time of inoculation and age at time of death, were independent of each other. The same conclusion resulted when the values for the male and female experimental groups were analyzed. Here too, there was lack of significance in the correlation coefficients (males: $r = +0.1911$, $P > 0.05$; females: $r = -0.0946$, $P > 0.05$). From this analysis it is concluded that within the time limits of this study, the age of the animal at inoculation had no influence on its age at death.

B. Mean Age at Death of Male and Female Experimental and Control Groups: In this section the age at inoculation has been eliminated from consideration because of the above findings, and the attained age at death of each animal was employed in

comparing the experimental with the control groups. The oldest male mouse in the group was a control animal which lived to be 923 days of age. The oldest female mouse was likewise a control, which died at the age of 968 days. Mean values and their standard errors were calculated for each of the four groups. These are shown in Table 10. The mean age at death of the 69 female syphilitic mice was 614.6±23.1 days. Their littermate nonsyphilitic controls died at a mean age of 694.9 ± 22.6 days. The difference of 80.3±32.3 days is significant. Considering the 71 male syphilitic mice, their mean age at death was 502.8±20.2 days, in comparison with a mean age of 591.7±25.4 days for their nonsyphilitic littermate controls. The difference of 88.9±32.4 days between these two values is also significant. The nonsyphilitic animals of both sexes thus lived longer than their syphilitic mates, and the observed differences in longevity were outside the realm of chance.

Cross comparisons between the sexes indicate other differences of interest. The female nonsyphilitic controls lived significantly longer than the male nonsyphilitic controls (difference = 103.2±30.3 days, $t = 3.03$, $P < 0.01$), and the female syphilitic mice lived to a longer mean age than did the male syphilitic mice (difference = 111.8±30.7 days, $t = 3.64$, $P < 0.01$). There was no significant difference between the mean age at death of the female syphilitic group in comparison with the male nonsyphilitic group (difference = 22.9 ± 34.3 days, $t = 0.67$, $P > 0.05$). These observations do not have the rigid controls that were operative in comparing the mean age at death of the experimental group with the control group of the same sex, for they contrast animals of different sexes, inoculated at different times with different inocula, and living in different cages. The observed differences do, however, have statistical validity.

These findings indicate that, under the conditions of the experiment, both male and female nonsyphilitic mice lived to a longer mean age than did their syphilitic littermates of the same sex. Further, the observations suggest that maleness has a detrimental effect on longevity, for both syphilitic and nonsyphilitic female mice lived to a significantly longer mean age than did the corresponding males.

C. Comparison of the Longevity of Individual Male and Female Syphilitic Mice with Their Nonsyphilitic Controls: The foregoing analysis has been concerned with group averages. An alternative method of analysis is available by virtue of the fact that each pair of mice was born on the same day and was later inoculated on the same day. Since these two time factors were constant for each pair, the length of life of each syphilitic mouse in a pair could be compared with that of its littermate control. In a previous section it was noted that five syphilitic mice, four males and one female, had been excluded because they had been killed for test of the persistence of infection. Since all these animals at the time of their death had outlived their controls, they are included in this analysis. With their inclusion there were 75 male pairs and 70 female pairs for comparison. Of the male pairs, 49 controls outlived their syphilitic littermates, or 65.3 ± 5.50% of the total. Twenty-four syphilitic mice lived longer than their littermate controls, or 32.0 ± 5.39% of the total. The death of both members of two pairs occurred on the same day. The difference between these values is significant (difference = 33.3 ±7.69%, $t = 4.33$, $P < 0.01$). A difference in the same direction was noted when the female pairs

were compared. In this instance, 47 of the 70 control mice, or 67.1 ± 5.62%, outlived their syphilitic littermates, and 22 syphilitic mice, 31.4 ± 5.55%, outlived their controls. Both members of a single pair died on the same day. The difference between these values is also significant (difference = 35.7 ± 7.90%, $t = 4.52$, $P < 0.01$). From this analysis it is evident that the proportion of control animals which outlived their syphilitic littermates was significantly greater than the proportion of syphilitic mice which lived longer than their nonsyphilitic controls.

TABLE 11.—*Distribution of Male and Female Syphilitic and Control Mice According to Age at Death*

	Females					
	Control Group			Syphilitic Group		
Age, Days	No.	Cumulative Total	Cumulative %	No.	Cumulative Total	Cumulative %
- 99	0	0	...	0	0	...
100-199	1	1	1.4	3	3	4.3
200-299	0	1	1.4	2	5	7.3
300-399	4	5	7.3	4	9	13.0
400-499	6	11	15.9	7	16	23.1
500-599	7	18	26.0	10	26	37.6
600-699	12	30	43.4	20	46	66.6
700-799	16	46	66.6	12	58	84.0
800-899	11	57	82.5	7	65	94.1
900+	12	69	100.0	4	69	100.0

	Males					
	Control Group			Syphilitic Group		
Age, Days	No.	Cumulative Total	Cumulative %	No.	Cumulative Total	Cumulative %
- 99	0	0	...	0	0	...
100-199	4	4	5.6	2	2	2.8
200-299	4	8	11.3	8	10	14.1
300-399	4	12	16.9	8	18	25.4
400-499	11	23	32.4	19	37	52.1
500-599	12	35	49.3	11	48	67.6
600-699	10	45	63.4	12	60	84.5
700-799	12	57	80.3	11	71	100.0
800-899	12	69	97.2	0	71	100.0
900+	2	71	100.0	0	71	100.0

D. Comparison of Distribution of Age at Death of Male and Female Syphilitic and Nonsyphilitic Mice: Still a third method for camparison of the longevity of the two groups is available. In Table 11 is shown the distribution of deaths by age in days, with cumulative totals and cumulative percentage. Of the females, 23 of the 69 syphilitic mice, or 33.3%, lived to be more than 700 days old, in contrast with 39 of the 69 controls, or 56.5%. The difference between these values is significant (chi-square = 7.48, $P < 0.01$). With regard to the male mice, 26 of the 71 control mice lived to be 700 or more days old, or 36.6%, while only 11, or 15.5%, of the syphilitic mice were 700 or more days old at death. The difference between these values is also significant (chi-square = 8.22, $P < 0.01$). This analysis indicates that fewer syphilitic than nonsyphilitic animals of both sexes reached age 700 days or over, and confirms the previous finding that the longevity of the syphilitic mice was not as great as was that of the nonsyphilitic animals.

COMMENT

A. *Changes Due to Syphilis.*—All animals were carefully observed during life and at death all were examined at autopsy. During life there was no variety of observed lesion or clinical sign in the syphilitic mice which was not also observed among the controls. All types of lesions observed at autopsy in the experimental group likewise had their counterparts in the control animals. There were no tissue changes which could be specifically diagnosed as syphilitic in origin.

B. *Causes of Death.*—Deaths resulted from a variety of causes, including pulmonary infection, gastroenteritis, hepatitis, tumors, particularly of the lymphoma type, urinary tract infections, myocardial fibrosis with heart failure, amyloidosis of kidneys, and prostatic hypertrophy with hydronephrosis. None of these caused death more frequently in one group than in the other. In many instances the cause of death could not be determined with certainty, but this was true to equal degree in both syphilitic and control groups.

C. *Persistence of Infection.*—Syphilitic infection in the mouse is asymptomatic. The only procedure by which it can be determined that a particular mouse harbors the infection is transfer of tissues from the suspect mouse to a rabbit, which, if the mouse is actually infected, will develop a dark-field-positive lesion. To have conducted this test on each mouse in the present study would have exposed them all to an operative procedure, the excision of a portion of skin.[18] This would have introduced a variable which in itself might have had an influence on the ultimate longevity of the animal. Since the scheme of the experiment was to reduce all variables to an absolute minimum, the surgical excision of skin for transfer to rabbits was not followed. Instead reliance was placed on a spot check of the persistence of infection in a random sample of mice which had been inoculated more than 600 days previously. As seen in Table 8, lymph nodes from each of five experimental mice produced dark-field-positive lesions in rabbits. The infection in these mice varied in duration from 642 to 872 days. On the basis of this 100% result, it is presumed that infection persisted in all the remaining mice under study. There is also indirect evidence of the persistence of infection; namely, the finding that the longevity of both male and female groups of experimental animals was reduced by approximately similar proportions when compared with the longevity of their nonsyphilitic littermates of the same sex.

D. *Longevity of Syphilitic Mice.*—Under the conditions of the experiment, both male and female mice with syphilis had a reduced lifespan when compared with their controls, and the differences were significant. Genetic and environmental factors were all constant, the only variable being the syphilitic or nonsyphilitic status of the animal. On the basis of these findings, it is concluded that syphilitic infection in mice, even though it produces no lesions of syphilis, in and of itself causes a reduction in longevity. The mechanism by which this result occurs is conjectural. Syphilitic animals died at an earlier age than nonsyphilitic controls of the same general pattern of diseases of which their nonsyphilitic mates later also died. From this it appears that syphilis in mice produces a lowered resistance to other diseases and acts as a general depressant of defense processes. If, as has been demonstrated, syphilis in the mouse does adversely influence longevity, we have here another

important bit of evidence for the conclusion that the mouse is actually infected and is not, as some have believed, merely a repository in which the inoculated treponemes are harbored in a viable state.

One cannot lightly dismiss the suggestion that depressed socioeconomic conditions are at least partly responsible for the high mortality rates among syphilitic persons. But over and above this consideration, there is the probability that in man, as in the mouse, syphilitic infection, by itself and exclusive of other modifying factor, reduces resistance to other diseases and shortens the lifespan. Seen in this lights, there is good justification for increasing rather than decreasing clinical, laboratory, and public health efforts to control the disease. The result of such a continued program will be many man-years of life saved. This should be the goal, and this will be the reward.

SUMMARY AND CONCLUSIONS

The evidence from vital statistics and clinical surveys indicates that syphilitic persons have a higher mortality than nonsyphilitic persons of similar age, sex and race.

Autopsy studies indicate that fewer syphilitic persons with lesions at autopsy survive the age of 70 than do nonsyphilitic persons in the same autopsy populations. Further, fewer syphilitic persons without specific lesions at autopsy live to be 70 or over than do nonsyphilitics in the same autopsy population.

Both syphilis prevalence rates and mortality rates from all causes vary according to socioeconomic factors; the better the socioeconomic conditions, the lower the rates.

It cannot be determined from studies of the human disease whether the decreased longevity of syphilitic persons results from syphilitic infection per se or from the socioeconomic conditions which foster high syphilis prevalence rates and high mortality rates from all causes.

In order to investigate the effect of syphilitic infection on longevity, under controlled conditions, a study of syphilis in the mouse was undertaken. A genetically homogeneous population of C57 black mice were employed. Littermate pairs of the same sex, one inoculated intraperitoneally with virulent treponemes, the other with saline solution, were housed throughout life in compartments of equal volume in a single cage. Feeding, watering, and environmental temperature were standardized. Sixty-nine pairs of females and 71 pairs of males were available for analysis.

The mean age at death of the control male and female groups was significantly greater than the mean age at death of the syphilitic groups of the same sex. The proportion of nonsyphilitic mice which outlived their syphilitic littermates was significantly greater than the proportion of syphilitic mice which outlived their control nonsyphilitic littermates. Significantly more control mice of either sex lived to be 700 days or older than did syphilitic mice of the same sex.

It is concluded that syphilitic infection in mice exerts a deleterious effect on longevity. Although the mechanism producing this effect is conjectural, it is suggested that syphilis depresses resistance to other mortal diseases. If the same mechanism is operative in man, and there is good reason to believe that it is, then syphilis by itself and exclusive of modifying socioeconomic factors exerts an adverse influence on the lifespan of the human host.

Miss Catharine L. Rowe and Mrs. Marion L. H. Freeman gave technical assistance in the conduct of the mouse experiments.

ABSTRACT OF DISCUSSION

Dr. NORMAN N. EPSTEIN, San Francisco: For many years Dr. Rosahn has been interested in the influence of syphilitic infection upon longevity. This problem does not lend itself readily to a satisfactory solution. The main difficulties lie in the interpretation of findings among the syphilitic and the nonsyphilitic populations. Clinical reports to date tend to disregard socio-economic factors among these persons. This fact may introduce a considerable error into the analysis of such clinincal data. In addition, the behavior of a syphilitic infection in mice, although comparable with human latent syphilis, cannot be accepted as being identical with the effects of the disease in human beings. Nevertheless, Dr. Rosahn has accumulated definite evidence from the study of clinical and postmortem material and by carefully planned experiments in mice that syphilis per se has an adverse influence upon the lifespan of human beings and mice.

He has demonstrated that human beings with latent syphilis, treated or untreated, in whom lesions of syphilis cannot be found, live a shorter time on the average than persons who have not had syphilis. Dr. Rosahn's findings indicate also that mice infected with syphilis, in which the disease is always asymptomatic, live a shorter time than uninfected mice. This concept is somewhat revolutionary, as most of us have felt that syphilis kills by destroying vital organs and tissues.

Other forms of stress have similar effects upon longevity, as indicated by the work of Lorenz (*Am. J. Roentgenol.* **63**:176, 1950) upon the lifespan of mice subjected to long-continued exposure to small amounts of radium radiation. In these experiments he demonstrated that the lifespan of mice is markedly reduced by daily exposures of 2.2, 4.4, and 8.8 r. Here, as in Dr. Rosahn's experiments the mice showed no lesions in their organs or tissues of radiation injury. Many other forms of long-continued stress undoubtedly act in a similar manner.

The author believes that a subject with a syphilitic infection is more susceptible to other morbid conditions than the uninfected subject. These in turn exert an adverse influence upon longevity. In other words, among the syphilitic population there will be more pneumonia, nephritis, arteriosclerosis, cardiovascular disease, etc., than among the nonsyphilitic population. This common belief among the clinicians appears frequently in the literature but still lacks uncontroversial scientific proof.

Another explanation for the shortening of the lifespan of syphilitics may be that the presence of T. pallidum in the tissues of the host over long periods produces a mild form of stress which calls into play the adaptation syndrome of Selye. This constant state of stress may lead to a slow exhaustion of this mechanism, so that these patients when subjected to added stress, such as infections and injuries, do not have the capacity to withstand them and thus succumb more readily.

I should like to ask Dr. Rosahn whether any evidence of these stress effects, such as a necrotizing type of arteriosclerosis or a reduction in size in the adrenal cortex or other lesions, were noted in his experimental animals. It would be very interesting to subject his animals to different forms of stress at varying periods to determine whether infected animals tolerate this added stress as well as the uninfected animals.

From a practical standpoint this paper makes one doubt whether our complacent attitude toward the patient with late latent syphilis is justified. In the past we have not been overly concerned with the prognosis in these patients, as long as they did not have syphilitic lesions of vital organs. It is quite likely that the disease exerts harmful effects upon the health of these patients in other and unknown ways.

Life insurance companies should be intensely interested in Dr. Rosahn's work and support further investigation in this field. They have consistently rated adequately treated syphilitics (without active lesions) from 135 to 200% normal standards risks because they have found that persons with a history of syphilis, even though adequately treated and negative serologically, actually have a life expectancy of 42% less than the normal standard expectancy (Impairment Study 1938, Compiled by the Actuarial Society of America and the Association of Medical Directors, New York, Actuarial Society of America, 1939).

DR. H. N. COLE, Cleveland: Dr. Rosahn, a pupil of the Syphilis Clinic at Johns Hopkins Hospital, and now at New Haven, Conn., has been working on this problem for many years. In 1946 he wrote an article, "The End Results of Untreated Syphilis" (*J. Ven. Dis. Inform.* **27**:293, 1946). I suppose it was, in part, in answer to the works of Boeck. The material was based on 3,907 autopsies, among which were 380 cases which had clinical, laboratory, or post-mortem evidence of syphilis. In 198 of these cases no treatment had been given for the disease outside of a few injections of bismuth or a few mercury rubs and potassium iodide. In but 38.9% were there any signs of syphilis. Death in 46 was primarily from syphilis and in 26 from cardiovascular syphilis. There were six cases of anatomical cerebral nervous system syphilis, a case of gumma of the brain, another of gumma of the heart, and three each of gumma of the liver and meninges.

He commented that if his findings represent a fairly accurate summary of end-results of untreated syphilis it may be concluded that had treatment been administered adequately to the entire group it could not possibly have effectively altered the course of the disease in more than 4 of 10 patients, since 6 of 10 untreated patients died with no evidence of syphilis, and only 2 of 10 actually died as a result of syphilis. Yet he concludes that syphilis is a dynamic disease, static at times, progressive and developing at others. With this we agree.

I like Dr. Rosahn's continued interest in syphilis. It is needed. Syphilis is still with us, and it is found to be more of a public health problem with war and its attendant aftermath of venereal complications. I congratulate the speaker for giving us further interesting, painfully procured data on the syphilis problem—all going to show that there are adverse influences of syphilitic infection on the longevity not only of man but of mice.

DR. STEPHEN ROTHMAN, Chicago: Dr. Rosahn used saline injections in his control animals. I should like to ask him whether he did not consider using extracts from normal rabbit testes for control injections rather than saline solution. After all, the experimental animals received a fair amount of foreign testicular protein with the spirochetal suspension. The conditions in the experimental animal would have been more closely simulated by giving the controls comparable amounts of testicular extracts.

I should also like to ask how we should interpret or define "latency" in the author's concept. Isn't it possible that specific microscopic changes are present in the small blood vessels throughout the vascular system without grossly visible lesions, and that these specific changes are the cause of decrease in longevity?

DR. ARTHUR G. SCHOCH, Dallas, Texas: I should like to ask Dr. Rosahn two questions: In one table presented it was shown that in litter and cage mates 65% of the syphilitic mice died before their mates, leaving 35% in which the reverse was true. Isn't this difference less marked than one would expect when one considers the other data?

My second question probably has occurred to Dr. Rosahn, and that is, would the difference in longevity of mice have been quite as marked if the mice inoculated with syphilis had received treatment? I realize full well that this would have interjected another variable into an almost pure experiment, but I should like to get Dr. Rosahn's impression of what the results might have been if treated syphilitic mice had been used in the experiment.

DR. MARTIN F. ENGMAN JR., St. Louis: If my father, Dr. M. F. Engman Sr., were here, I believe he would discuss this paper. I think he would feel as I do that this is a well-thought-out piece of work. There is one aspect of this problem that has not been mentioned and that is the special type of person who is a syphilitic. Certain types of persons seem to have been born to have certain problems in their lives. For instance, in a magazine some time ago an article by someone in the insurance field appeared with the title "Accidents Go on Happening to the Same People." The author stated that there is an "accident-prone" type of person.

When I first went into practice, my father would discuss with me the syphilitic person. He is the type of person who, through some inherent lack of judgment, acquires syphilis in the first place. This type of person does not make a good patient. He is careless about his treatment; he does not pay his bills, and he is difficult to handle. This type of person probably is not one whose life expectancy will be as good as the average anyway because of his inherent poor judgment, carelessness about himself, and lack of responsibility. This factor is very difficult to evaluate, but I think it should be taken into consideration.

DR. FRED D. WEIDMAN, Philadelphia: Dr. Engman has indicated one upsetting item in the analysis of the statistics in the case. It prompts me to bring up one other, namely, the autopsy material. I have had considerable experience at the autopsy table and know how variable the value is. Autopsies are not as thorough, for example, during the summertime, when the humidity is high, as they are during the winter and when the teaching season is "on."

Commonly, at least in the Philadelphia area, the brain and the spinal cord are not examined; it is a more or less laborious task, even though one has a prosector to do it. Perhaps Dr. Rosahn has covered this point in his paper, but I would judge that those autopsy reports in which there was not a complete examination of the body should not have a place in a paper with such a heavy statistical basis.

DR. PAUL D. ROSAHN, New Britain, Conn.: Dr. Epstein mentioned the theoretical possibility that the stress syndrome may be the responsible factor in producing the results noted in this study, and I have had the impression that this is probably true. Some indirect pieces of evidence indicate that perhaps the answer does lie in this direction. In the first place, the subject with active syphilis has a marked increase in eosinophiles in the peripheral blood, and the eosinophiles remain relatively elevated even though activity of the disease has disappeared. Secondly, there is a depression in the lymphocytes, which increase in the peripheral blood as activity disappears. There is perhaps a relation between eosinophile and lymphocyte levels and the activity of the adrenal gland. It would be highly desirable to initiate an experiment to determine whether the adrenal gland is actually implicated in this general problem. I think Dr. Epstein has made an extremely important suggestion, and I want to thank him for it.

Dr. Cole referred to the work of Bruusgaard and my review of autopsy material on untreated syphilis which was published several years ago. The remarkable fact about the autopsy material is that the findings parallel almost exactly the observations of Bruusgaard based upon Boeck's cases. The parallelism between the clinical studies of Bruusgaard and the autopsy findings in New Haven suggests that the two results support each other. Whether or not lack of treatment produced the profound effect that I demonstrated I do not know, because I did not have a similar group of adequately treated patients for comparison, but the fact is that in this material 2 of 10 persons with syphilis died directly as a result of syphilitic disease as indicated by anatomical evidence of syphilis at autopsy.

Dr. Rothman has raised two extremely interesting questions, the first one suggesting that the control animals might better have been inoculated with testicular extract. The syphilitic material which was inoculated into these mice was not normal testicular material. It was the cartilaginous type of syphiloma which is analogous to the hard indurated primary chancre. It contained no evidence of testicular substance, and, therefore, testicular tissue was not used in the control inocula. Rr. Rothman raises the question of the interpretation of tissue lesions in terms of anatomical latency. I must confess that I have never been able to satisfy myself that I can make a diagnosis of latent syphilis on the basis of tissue examination. Perhaps there are morphologic changes in latent syphilis with which I am not familiar, but I am not certain that I can make such a diagnosis.

Dr. Schoch noted that 65% of the uninfected mice outlived the infected partner, and he asks whether this figure is lower than I had expected. I think it was lower than I had expected. I thought that a larger proportion of the controls would outlive their syphilitic mates than actually was found, and I cannot account for the low figure which Dr. Schoch mentioned. Dr. Schoch asked also whether a difference would have been so manifest if treatment had been given. I think that this question requires a philosophical reply, since we have no factual material upon which to base an answer. I believe that if treatment had been given the results would probably have been no different from those reported. We would have been treating latency, and it is questionable in my mind whether the host reactions in latent syphilis are affected by treatment. This is a personal position in support of which I cannot cite any experimental data. Unfortunately, we were unable to include a third group of treated mice in our experiment.

Dr. Engman raises the question of the environment of the syphilitic and asks whether the syphilitic is the accident-prone type of person. This is the concept that I have had, and one I am sure most of us here have had in the past. It was exactly for the purpose of testing this

concept that the mouse experiment was designed. If we can draw a parallel between the mouse experiment and human material, we have eliminated the accident-prone type of subject in the mouse experiment. Still, the differences were as noted; the syphilitic did not live as long as the nonsyphilitic. Thus, even if in human material we do consider that there are syphilitics who are of the accident-prone type and even though these persons by virtue of their proneness to accidents do not live as long as others, still there is a residue of persons whose longevity is decreased because they have syphilis, and not solely because they are of the accident-prone type.

Dr. Weidman presents a most interesting statement in terms of the validity of the autopsy material, and I can assure Dr. Weidman that I was well aware of the criticisms which he has made. I compared those in whom the brain and spinal cord were not examined with the groups in whom these organs were scrutinized. There were no significant differences between these two groups with respect to the variables which I was investigating, and therefore I ventured to include in the final material those persons in whom the brain and spinal cord were not examined.

ANNULAR CONSTRICTIONS OF THE DIGITS

Presentation of an Interesting Example

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AT THE 1950 meeting of the Atlantic Dermatologic Conference, one of us (R. C. V. R.) presented a 6-month-old Negro child with annular constrictions of several fingers and toes. During the discussion which followed the presentation of patients, several physicians remarked on the resemblance of the constrictions to ainhum, a condition usually considered peculiar to persons in tropical countries. None recalled seeing a similar condition in such a young child.

During one of the child's follow-up visits to the Family Syphilis Clinic, two junior staff members remarked that they did not consider this constriction a rare entity, having seen several similar cases in plastic surgical clinics in other hospitals. Although a search of the records failed to reveal any other hospital admissions for such a condition, a nurse who had been associated with our hospital for many years stated that she had seen several patients with "ring fingers" in our own plastic surgical clinic.

Stimulated by the mixed reaction to this child's abnormalities, we decided to investigate the literature. Our results warrant the following case report and brief review of the subject.

REPORT OF CASE

D. A. (J. H. H. A-72963), a Negro girl born Aug. 14, 1949, was brought to the Family Syphilis Clinic of The Johns Hopkins Hospital at 6 weeks of age because her mother had been treated with penicillin for secondary syphilis prior to pregnancy.

There was no history of birth trauma. The mother stated that the infant seemed normal except for "bands" around several fingers and toes.

Physical examination revealed no abnormal findings except annular constrictions as illustrated in the photographs (Figs. 1 and 2). These were considered as ainhum-like deformities.

Roentgenograms showed soft tissue constrictions of the proximal phalanges of the left index finger and right ring finger. No bony changes were noted.

There seemed to be no impairment of circulation, and the patient was referred to the plastic surgery clinic, where it was decided to do nothing until she was one year of age. At that time, multiple incisions were made in the constrictions, and on June 20, 1951, nine months later, the bands were noted to be less deep, and it was felt that a good result had been obtained.

From the Family Syphilis Clinic of the Venereal Disease Division of the Medical Clinic of the Johns Hopkins University and Hospital.

Fig. 1.—Annular constrictions of the fingers.

Fig. 2.—Annular constrictions of the toes.

Repeated serologic tests for syphilis elicited negative reactions over a 15-month period, and the child is considered to be nonsyphilitic.

COMMENT

Annular constrictions of the extremities may be divided into four general classes, etiologically.

Traumas, such as encircling lacerations, burns, or frostbite, frequently heal with band-like scars. Since the primary cause of such a lesion is usually obvious, further discussion is not indicated.

Much more important, because of confusion in differential diagnosis, is ainhum. This disease of obscure causation [1] is usually considered limited to tropical and subtropical climates and is seen almost entirely in dark-skinned persons. Usually the fifth toes are involved by a painless, progressively constricting band, distal to which the blood vessels are thickened and enlarged but the caliber is narrowed by endarteritis.[2] The degenerative process is both fibrous and fatty. The end-result is spontaneous amputation of the distal portion of the digit. There is no known medical treatment, and prophylactic surgery is rarely of benefit.

Constricting bands associated with other diseases may be confused with ainhum. Leprosy,[3] scleroderma,[4] diabetes,[5] syringomyelia, and pityriasis rubra pilaris [6] and hyperkeratosis of palms and soles [7] have each been described on one or more occasions as forerunners of or coexisting with ainhum or an ainhum-like condition.

The signs presented by our patient fit most closely the final group of annular bands, which are congenital in origin. These are present at birth, or are noticed in the early neonatal period, and vary in severity from simple, easily correctable grooves, such as on this little girl, to complete amputation of a digit or limb below the constriction. The patients described by Bluefarb [8] and Johnson [9] presented conditions which fall into this category of congenital anomalies.

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8. Bluefarb, S. M.: Constriction of Finger Simulating Ainhum: Report of Congenital Case, Arch. Dermat. & Syph. **57**:741, 1948.

9. Johnson, H. M.: Congenital Cicatrizing Bands: Report of Case with Etiologic Observations, Am. J. Surg. **52**:498, 1941.

SUMMARY

A Negro infant with annular constrictions of several digits was presented at a large dermatological meeting, and the condition was not recognized.

This, or a similar condition, is a clinical entity known to the plastic surgeon and obstetrician and, since it involves the integument, deserves recognition by the dermatologist.

There are four etiological groups of annular bands: those definitely related to trauma; true ainhum, usually considered limited to dark-skinned persons of the tropics or near-tropics; bands due to other diseases which simulate or occur concomitantly with ainhum, and congenital bands, an example of which is presented here.

IN VITRO FUNGISTATIC EFFECT OF TETRACHLOROPARABENZOQUINONE

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ATLANTA

TETRACHLOROPARABENZOQUINONE (TCPB), marketed under the trade name "spergon," has been in general use for the prevention of plant diseases, such as seed decay and "damping-off," since 1940, when it was discovered by Cunningham and Sharvelle [1] to be an excellent seed protectant against phytopathogenic fungi. It is a yellow crystalline solid, insoluble in water, an aqueous suspension of which assumes a violet color resulting from a gradual hydrolysis to chloranilic acid.

In 1946 Collins, McGavack, and Boyd [2] reported laboratory trials of tetrachloro-*p*-benzoquinone as a fungistatic agent against human pathogens. Serial dilutions of the compound in "nutrient dextrose agar broth" were inoculated with strains of Microsporum canis (Microsporum lanosum; Microsporum felineum), Microsporum audouini, and Trichophyton tonsurans. Minimum and maximum fungistatic concentrations were found to be 0.8% and 3.0% respectively, 8 of the 29 strains used being inhibited by 1.5%. In clinical tests, 169 patients with tinea capitis apparently caused by Microsporum species were treated with ointments containing 0.5 to 3.5% tetrachloro-*p*-benzoquinone. Seventy per cent of these were reported to "have obtained relief," and 30% were apparently cured. The authors stated that the drug was "relatively non-toxic to rats and rabbits in fungicidal concentrations." They concluded that "the fungicidal activity of tetra-chloro-para-benzoquinone apparently depends upon the loosely combined chlorine atoms, which are relatively stable in acid media, but readily soluble and active in the presence of alkalinity."

Kligman and Rosensweig,[3] in 1948, in studies on a series of new fungistatic agents, tested tetrachloro-*p*-benzoquinone in vitro by the paper disc method against Trichophyton mentagrophytes and M. canis (lanosum), and by a dilution method against M. audouini, Trichophyton rubrum, and Epidermophyton floccosum. Tests with the first two organisms resulted in 13 and 19 mm. of inhibition, respectively, at a drug concentration of 1 : 1,000, but none at 1 : 5,000 or lower. By the dilution pro-

From the Communicable Disease Center, Public Health Service, Federal Security Agency.

1. Cunningham, H. S., and Sharvelle, E. G.: Organic Seed Protectants for Lima Beans, Phytopathology **30**:4, 1940.

2. Collins, J. G.; McGavack, T. H., and Boyd, L. J.: The Effect of Tetra-Chloro-Para-Benzoquinone upon Human Fungus Infections: In Vitro and in Vivo Studies with Tinea Capitis, Urol. & Cutan. Rev. **50**:276, 1946.

3. Kligman, A. M., and Rosensweig, W.: Studies with New Fungistatic Agents: I. For the Treatment of Superficial Mycoses, J. Invest. Dermat. **10**:59, 1948.

cedure, the drug had a minimum fungistatic concentration of 0.1% against each of the three organisms. These authors also performed toxicity tests, which indicated that tetrachloro-*p*-benzoquinone is relatively nontoxic to mice when injected intraperitoneally and is nonirritating to the rabbit conjunctival sac.

McGavack and associates,[4] in 1949, using tetrachloro-*p*-benzoquinone as an ointment in various bases in concentrations of 5 to 50%, as well as in powder form, concluded that the majority of dermatophytoses, except those due to M. audouini, could be cured by a 10% concentration of the compound in a specific ointment base (oil-in-water emulsion) but that for M. audouini infections a strength of at least 30% is needed, with 50% indicated if the lower concentration is ineffective.

It had been reported by Hatch,[5] in 1945, in tests on 15 dogs, that "spergon," alone or in combination with equal proportions of boric acid or starch, gave very promising results in the control of moist summer eczema, believed to be partially or entirely due to Alternaria tenuis. Allen,[6] however, stated that experimental studies on dogs lent no support to the opinion that A. tenuis is implicated in the causation of the disease.

EXPERIMENTAL DATA

The present in vitro experiments were undertaken at the suggestion of local dermatologists who conducted clinical trials on a large series of patients with tinea capitis. Results of some of the clinical studies have been published elsewhere.[7] No attempt is made here to evaluate the therapeutic efficiency of tetrachloro-*p*-benzoquinone, since it is well known that the clinical effectiveness of a compound depends upon many factors, some of which are unrelated to its in vitro fungistatic activity.

The organisms tested, including both stock cultures carried in this laboratory and freshly isolated strains, were dermatophytes of the following species: M. canis, Microsporum gypseum, M. audouini, T. rubrum, T. mentagrophytes, and E. floccosum. Commercial "spergon," which is 99% tetrachloro-*p*-benzoquinone, was employed in the early experiments. Later, pure tetrachloro-*p*-benzoquinone was used.[8]

Fungistatic tests were conducted according to three distinct procedures.

A. *Broth Dilution Method.*—Five concentrations of "spergon" (0.004, 0.02, 0.04, 0.1, and 0.4%) in Sabouraud dextrose broth were prepared in triplicate in 125 ml. Erlenmeyer flasks, 25 ml. per flask. The broth alone was used as a control. The flasks were autoclaved for 10 minutes at 15 lb. (6.8 kg.) pressure. It was noted that the powder had not dissolved completely when in concentrations of 0.04% and above. Each flask was then inoculated with a pinpoint piece of mycelium from a slant culture of M. audouini 54 (freshly isolated) and all were incubated in the dark at 25 C. for one month.

4. McGavack, T. H.; Weiner, D.; Lo Cascio, A.; Bell, M., and Boyd, L. J.: Influence of Tetra-Chloro-Para-Benzoquinone on Human Fungous Infections, Arch. Dermat. & Syph. **59**:94, 1949.

5. Hatch, R. D.: Spergon—Fungicide for Summer Eczema in Dogs, J. Am. Vet. M. A. **106**:278, 1945.

6. Allen, T.: Some Observations on the Relationship of Alternaria Tenuis to Canine Eczema, J. Am. Vet. M. A. **106**:163, 1945.

7. Rankin, J. L.; Dobes, W. L.; Jones, J. W., and Alden, H. S.: Ringworm of the Scalp: Treatment with Tetrachloro-Para-Benzoquinone, South. M. J. **44**:616, 1951.

8. Dr. T. W. Brasfield, of the Naugatuck Division of the United States Rubber Company, donated "spergon" and tetrachloro-*p*-benzoquinone.

Results are given in Table 1.

The above procedure was repeated with pure tetrachloro-*p*-benzoquinone and the following organisms: M. canis 367, M. gypseum A81 and 213, M. audouini A150, T. rubrum A149, T. mentagrophytes 278, and E. floccosum 118 (all stock strains).

The results of these tests, read after 14 days, are also presented in Table 1. Tetrachloro-*p*-benzoquinone was found to exert a considerable effect on the pH of the uninoculated broth, values after autoclaving ranging from pH 4.6 in the 0.4% flasks to 6.4 at 0.004%, while the Sabouraud controls had a reading of 6.6. How-

TABLE 1.—*Fungistatic Activity of Tetrachloroparabenzoquinone by Broth Dilution Method*

	Amount of Growth After 2 Weeks *					
	Concentration (%) of TCPB in Sabouraud Broth					
Organism	0	0.004	0.02	0.04	0.1	0.4
Microsporum canis 367	++++	++++	+	0	0	0
M. gypseum A81	++++	++++	+	±	0	0
M. gypseum 213	++++	++++	++++	+	0	0
M. audouini A150	++++	++++	+	0	0	0
M. audouini 54 †	++++	++++	++++	0	0	0
Trichophyton rubrum A149	++++	++++	0	0	0	0
T. mentagrophytes 278	++++	++++	+	0	0	0
Epidermophyton floccosum 118	++++	++++	0	0	0	0

* Average of three flasks. M. audouini 54 read at one month.
† "Spergon" used instead of pure TCPB.

TABLE 2.—*Fungistatic Activity of Tetrachloroparabenzoquinone by Agar Cup Method*

	Diameter of Inhibition Zone (Mm.) (Average of 2 Tests)					
	Concentration (%) of TCPB in Acetone					
Organism	0.1	0.05	0.025	0.0125	0.0062	Acetone Control
Microsporum canis 367	16	12	0	0	0	0
M. gypseum A207	24	0	0	0	0	0
M. audouini 2241	19	14	9	0	0	0
Trichophyton rubrum 1986	25	17	14	10	0	0
T. mentagrophytes A143	22	16	10	0	0	0
Epidermophyton floccosum A334	28	20	12	10	0	0

ever, auxiliary tests indicated that the fungistatic effect of the chemical was independent of pH, at least in the acid range.

B. *Agar Cup Diffusion Method.*—Duplicate thick pour plates of the following dermatophytes were prepared in Sabouraud dextrose agar: M. canis 367, M. gypseum A207, T. mentagrophytes A143, E. floccosum A334 (all stock strains), M. audouini 2241, and T. rubrum 1986 (both newly isolated). By means of a sterile cork borer 8 mm. in diameter there were removed from each plate six plugs of agar, so that one cup was left in the center and five spaced equidistantly about it. Into the central cup was pipetted 10 drops of acetone, while the remaining cups were filled with acetone solutions of tetrachloro-*p*-benzoquinone in concentrations of 0.1, 0.05, 0.025, 0.0125, and 0.0062%. The plates were incubated at 25 C. and read after six days.

The results are shown in Table 2.

C. *Agar Slant Method.*—A series of experiments was conducted in which tetrachloro-*p*-benzoquinone dissolved in 95% ethyl alcohol was incorporated into Sabouraud agar slants

in graded concentrations and the media adjusted to uniformity of pH. In the higher concentrations the drug precipitated as soon as it was added to the melted agar, but the resulting suspensions observed upon solidification of the media were finely particulate and uniform. A pinpoint-sized inoculum of mycelium was placed in the center of each slant, and the cultures, in triplicate, were incubated at 25 C. for periods of two to four weeks. Controls consisted of various concentrations of alcohol in Sabouraud agar. Various strains of all six of the dermatophyte species previously mentioned were used at different times.

Results were not consistent, with even the alcohol controls varying from time to time in their fungistatic action. However, it was found that in any one experiment identical results were obtained with a given concentration of tetrachloro-*p*-benzoquinone tested at an initial pH of either 6.0 or 8.0, thus indicating that the compound is equally active in vitro on both sides of neutrality. Further, it was demonstrated again that, in general, inhibition of growth of the test organisms is proportional to the concentration of tetrachloro-*p*-benzoquinone and that this compound is effective in relatively minute concentrations.

SUMMARY

Tetrachloro-*p*-benzoquinone has been shown by three different fungistatic tests to be an efficient fungistatic agent in vitro against the commoner dermatophytes. Several strains of six species in three genera were employed in the experiments, all being found to be sensitive to small concentrations of the drug.

DELUSION OF PARASITOSIS (ACAROPHOBIA)

Further Observations in Clinical Practice

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LOS ANGELES

IN THE year 1946 a discussion of the delusion of parasitosis was presented in the dermatologic literature [1] under joint authorship with the late Dr. Hiram E. Miller, in which it was pointed out that patients who unalterably but falsely believe that the skin is infested with parasites are suffering from a severe mental illness only rarely correctable by the dermatologist and occasionally by the psychiatrist. The designation "acarophobia," under which the syndrome was previously classed, was deemed inappropriate because such patients rarely selected the organism of scabies as the infesting parasite and seldom exhibited any of the fear, dread, or hysteria usually associated with the other phobias. Only 45 cases could be found recorded in the literature, whereas the disorder occurs much more frequently than that figure would indicate. Six cases which had come under our own observation were presented and discussed.

In reviewing and tabulating the features recorded, it was believed that all cases could be classified in one or another of four psychiatric categories: toxic psychosis, the paranoid type of dementia praecox, involutional melancholia, and paranoia (including the paranoid states often observed in arteriosclerosis, syphilis, alcoholism, and senility).

Since that paper was completed, I have had the opportunity of observing 16 new cases in which the delusion of parasitosis was prominently displayed. The details concerning 18 additional cases have been related to me by dermatologists and psychiatrists in the immediate geographic locality. This paper is intended to present evidence to confirm many of our previous opinions, to revise a few, and to attempt to integrate the symptom complex more instructively into the concept of the psychosomatic dermatoses.

TERMINOLOGY

To illustrate the reasons for preferring the term "delusion of parasitosis" for most cases, it is appropriate to cite the history of one patient who was recently observed, and who was the first to be encountered either in the material previously reported or that assembled subsequently in which the disorder could be accurately designated as "acarophobia."

CASE 1.—A 32-year-old rather effeminate man working as a bank teller stated that he had had scabies seven years previously. This had been diagnosed by a competent physician, and he had been shown the living Sarcoptes under the microscope by him. The patient believed the

From the Department of Dermatology, University of Southern California School of Medicine and the Los Angeles County General Hospital.

1. Wilson, J. W., and Miller, H. E.: Delusion of Parasitosis (Acarophobia), Arch. Dermat. & Syph. **54**:39-56 (July) 1946.

infestation had been acquired by shaking hands. He admitted that he had obtained a complete cure within two weeks, but on several occasions since then he had consulted a physician because he thought it possible that he had again acquired scabies, even though for the past five years he had refrained from shaking hands as well as from all other physical contact with other persons or animals. He admitted that findings on such examinations had been negative at all times, but he "felt better if he had himself checked up." He had acquired the habit of washing his hands frequently, perhaps 30 times a day, to avoid infestation. Recently he had begun to notice the condition of the hands of his clients at his bank window, and if any indication of a skin disease seemed evident he felt compelled to wash his hands immediately after receiving money or a check. He also had developed the habit of placing such money to one side and exchanging it on some pretext with a neighboring clerk, so that he himself was in no further danger of handling it. He was particularly worried during the last few days because one of his fellow workers had noticed and speculated upon the undue frequency with which such exchanges occurred. A suggestion that psychiatric consultation might be helpful met with the response that "all that was needed was periodic examinations to exclude the possibility of infestation."

This man actually possessed a morbid fear of acquiring scabies, and he apparently had no persistent delusion of actual infestation; although he thought it possible on occasion, he was always willing to accept the physician's dictum that none was present. This case should not be classed as "delusion of parasitosis"; there was present probably no more than an obsessive-compulsive neurosis, expressed as acarophobia.

It is thus apparent that the term acarophobia cannot be entirely discarded although it will be applicable only rarely. Of course, it is difficult at times to decide whether a patient exhibits a phobia or a delusion; sometimes both are present. Patients suffering from a phobia uncomplicated by delusions usually can be successfully reassured by appropriate examinations (for a while at least) that no actual disease is present.

Some of the patients usually classed as possessing syphilophobia and other phobias undoubtedly present, instead, a persistent delusion that the disease actively is present, and the more appropriate term, "delusion of parasitosis" should be substituted. The following case report will serve as an example.

CASE 2.—A 60-year-old housewife stated that she had acquired syphilis in 1917, from her first husband, from whom she was shortly thereafter divorced. The doctor who examined her assured her that "all of the tests for syphilis were negative." Another physician was immediately consulted and again all tests, including those performed on the cerebrospinal fluid, gave negative results. There had been no genital or oral lesions, and no cutaneous eruption had been observed at any time.

In spite of this evidence, the patient insisted so strenuously that she needed treatment for syphilis that a course of 15 injections of arsphenamine was administered. Treatment was again obtained during the succeeding year, after which the patient "felt well until 1929, when the disease gradually returned." Alternating courses of neoarsphenamine and bismuth injections were administered for one and one-half years, during which time "all tests" were admittedly negative. The disease "almost disappeared but never entirely"; she was able to "stand it" during the next 11 years by seeking and receiving a "few shots now and then." Since 1942, however, it has been her habit to "take a short course of bismuth every six months, perhaps 10 or 12 injections, to keep the symptoms down."

At the time of my examination she complained of some troublesome symptom in virtually every part of her body. Except for a moderately enlarged heart, obesity, and tenderness wherever palpated, physical examination was entirely normal. Serologic tests for syphilis gave negative results. She assured me that the "tests didn't work in my case, but the disease was there just the same."

Considerable effort was made to convince her that treatment for syphilis was unnecessary, but she is still receiving "a few shots of bismuth every six months."

It is evident that for over 30 years this patient has possessed an unshakable delusion that syphilis has been present. She affirms that she is "not afraid of it—it will do no harm if she takes her treatments." To call this a case of "syphilophobia" seems less accurate than to use the designation "delusion of syphilitic infection" or of "parasitosis."

INCIDENCE AND IMPORTANCE

The fact that 34 instances of the delusion of parasitosis occurring in one locality have been brought to the attention of one observer in seven years proves that the condition is not rare. Moreover, most of these patients exhibited fully developed persistent delusions, which had been present for from several months to several years; undoubtedly many additional cases, similar but less spectacular or of shorter duration, must have occurred in the same geographic region during the same period. Since the appearance of such a delusion may be the earliest recognizable reliable sign that a person previously classed as "nervous" is actually beginning to suffer more profound psychologic change, and since psychiatric care is in most instances much more effective if it is utilized in the early stages of the disease, it is important that all practitioners of medicine be able to recognize this syndrome, its significance, and the necessity of at least attempting to furnish its possessors adequate psychiatric consultation without delay.

PSYCHIATRIC DIAGNOSIS AND CLASSIFICATION

In studying the new material it has become apparent that revisions should be made in some of the statements regarding psychiatric diagnosis and classification contained in the article previously referred to.[1] At that time it was believed that all cases of the delusion of parasitosis could be placed in one or another of four psychiatric categories, each representing a true major psychosis. However, at least two cases have recently been observed in which the delusion was apparently well developed in persons whose mental aberrations almost certainly were no more than psychoneuroses; the facts presented warrant additional discussion.

The dividing line between the true psychoses and the psychoneuroses is not easily discernible except to the physician trained in psychiatry and even to him only after considerable detailed study in many instances. Our previous attempt to limit the syndrome to the true psychoses was based upon the concept that the psychoneurotic individual is always in contact with reality while the psychotic patient is characteristically at some time and in some way out of such contact. To us an unshakable delusion represented a loss of contact with reality and marked the possessor as suffering from a true psychosis. However, further study and conversations with competent psychiatrists have led me to believe that such a delusion as this may at times be exhibited by a psychoneurotic individual for many years—in fact, as long as the intolerable situation which is fundamentally responsible for the psychoneurosis remains unchanged. Such an individual rarely becomes truly psychotic; in fact, it is usually accepted that he is singularly immune from such a change, although he may deteriorate to such an extent that he becomes totally unable to care for himself and his life in many ways is more disturbed than that of the average truly psychotic patient.

Furthermore, paranoid trends are encountered at times in almost all classes of psychologic disorders; the adjective paranoid, by definition, indicates simply

the "presence of systematized delusions which are built up in logical form." Frequent use is made by psychiatrists of such classifications as paranoid type of schizophrenia (dementia praecox), paranoid type of involutional melancholia psychosis, paranoid type of manic-depressive psychosis, paranoid type of toxic psychosis, as well as "true paranoia"; less frequently, paranoid symptoms are recognized in other disorders. Indeed, the inability to recognize and admit his own faults, defects, and delusions characterizes a person as having a "paranoid personality." It seems likely that if sufficient case material could be studied, occasional examples of the delusion of parasitosis would be found in persons exhibiting a wide variety of mental aberrations.

In the disease under discussion it is apparent also that the opportunity for prolonged study usually necessary to assemble sufficient details to assign a case to the proper psychiatric classification will seldom be afforded. In many of the cases tabulated in our previous paper characteristics sufficient to allow accurate classification to be made were lacking, and only 5 of 34 recent cases are known to have had adequate psychiatric study. Patients with delusions such as these resist any and all attempts to cause the delusions to disappear; the merest hint that a psychiatrist would be the proper person from whom to seek help will frequently terminate the interviews. Moreover, they seldom become sufficiently disturbing to others in the community to warrant forcible commitment to psychiatric institutions, where study would be obtained.

It is apparent that further attempts to classify cases of the delusion of parasitosis into definite psychiatric entities on the basis of the fragmentary information usually elicited by the dermatologist untrained in psychiatry are hazardous and difficult and can serve but little additional useful purpose either to him or to his patient. It will probably be sufficient to reiterate that this symptom-complex indicates the presence of a mental aberration of a severer degree than that which can be handled adequately by the superficial type of psychotherapy which the physician trained only in dermatology is able to offer his patient and that psychiatric consultation should be recommended.

It seems wise, therefore, at this time to hold in abeyance any further discussion of psychiatric classification as well as any attempts to tabulate and draw statistical conclusions from the new case material at hand. A few interesting case variations will be recorded here and tentative conclusions drawn where they seem to be indicated.

DISCUSSION OF CASES

It is interesting that four of the patients recently observed attributed the cause of the so-called infestation to precisely the same sequence of events. During and immediately after World War II, the shortage of housing in Los Angeles caused it to be considered unpatriotic for one or two persons occupying a large home not to share it with defense workers or military personnel. Three women who yielded to this pressure by allowing other families to move into their homes subsequently manifested the delusion of parasitosis; the husband of one of them also became afflicted. These people were torn between the dread of appearing to be unpatriotic and that of being deprived of their accustomed privacy and seclusion. In all three instances it was asserted that the unwanted tenants had brought "bugs" with them, and this belief was made to serve as sufficient reason for eviction. Two of these instances seem worthy of recording.

CASE 3.—A 34-year-old woman had lost a child of 3 years through an accident 10 years previously. Although the circumstances did not warrant it, she was convinced that her own incompetence as a parent had been the sole cause. Complete sexual frigidity was said to have developed, but two illegal abortions had been necessary to protect her from again assuming parental responsibility. She had been subject to headaches and attacks of dizziness and vomiting at frequent intervals when under even moderate stress. Her husband was very devoted to her and catered to her every wish.

Several months previously her nephew had obtained a position in a defense industry in the city in which the patient lived, but owing to the wartime housing shortage he was unable to find a home for his wife and two young children. The patient had permitted this family to move into her home because she believed it to be her patriotic duty to do so, although the resulting noise had markedly increased her headaches and dizziness.

After a few months she began to complain of constant generalized pruritus, which she stated was caused by parasites brought into the home by her nephew's unclean children. She insisted that the children be scrubbed frequently, sometimes several times daily, and she herself bathed almost incessantly. She had applied benzyl benzoate in lotion form over her entire body each day for over two months; her husband and the tenants were subjected to this medication on several occasions.

She presented a diffuse, scaly, erythematous dermatitis considerably less severe than might be expected from such strenuous medication. There was no evidence of any other disease capable of causing the pruritus, and no parasites could be discovered.

The patient stated that she could not bring in any of the insects as requested; although she said she "could see them," she had been unable to "catch any of them because they were too fast." She explained the fact that no one else living in the household suffered any itching by saying that the insects "liked her tender blonde skin" much better than that of her darker-complexioned companions.

Arrangements were finally made to house the unwanted family elsewhere, and the patient's pruritus promptly ceased; the delusion disappeared as well. Seven months later she stated that she had remained free of further similar trouble.

In this case the delusion apparently developed after it became evident that the complaint of the increase in her headaches and dizziness would not be successful in causing the unwanted guests to depart. It was also necessary for her to have a more convincing reason both for herself and for others to explain her lack of patriotism in not continuing to contribute part of her home to the war effort.

Psychiatric consultation was not obtained. The patient's husband believed her to be physically instead of mentally ill and was willing to accept the fact that actual parasites were annoying her, although he himself had never seen them nor had any symptoms of infestation. It was considered possible that the entire episode was on the patient's part a mere sham, intentionally acted out to satisfy a desire; this suggestion to the patient's husband met with heated denial. Opportunity for further study was precluded by the cessation of the delusion, and without more information psychiatric classification is probably inaccurate. The case seems, however, to be one of psychoneurosis.

CASE 4.—A 40-year-old woman stated that she and her husband had been well and happy until three months previously, when a soldier and his family were allowed to move into the house. They "brought many bugs with them in their luggage." The resulting turmoil was too much for the visitors, who moved out within a month, leaving some articles, including clothing, to be obtained later. The patient continued to observe "tiny bugs of some sort looking like black-gray dots and numerous beetle-type bugs much larger than the others." Since that time every day had been spent entirely in attempts to rid the house and the bodies of the patient and her husband of the "parasites," but both remained firmly convinced that they were still present. Special attention was given to the departed tenants' belongings, from which a constant stream of insects was said to emanate. She stated that "every stitch of clothing and bedding we own has been boiled and changed each day; only paper towels and tablecloths have been used and

immediately burned." She exhibited an unusual localization of the worst of her discomfort to her "left upper arm and right big toe." She stated that she could see the "little black dots come out of her skin by looking through the magnifying glass." When each bug emerged she claimed to experience a stinging sensation. The husband complained of intense itching and a "feeling of gravel under the skin." He, too, was possessed of an unshakable delusion that parasites were present. Several physicians, including two dermatologists, had been visited and various diagnoses obtained; twice the patients had been told they were infested with scabies, once with "bird lice." Both patients admitted that until that time there had been some doubt in their minds as to the presence of any actual infestation; after that they were firmly convinced.

The woman was given six vials; two days later she returned them all filled with bits of debris of all sorts which she asserted were "myriads of the bugs." These were examined, and she was informed that no parasites were present. Her response was that which has come to be the expected one; she "simply couldn't understand how they all could get away!" There was not the slightest doubt in her mind that she had actually placed the bugs in the vials originally. She was strongly advised to stop all medication directed against parasites. She reported in one week that she had been able to refrain from medication for three days only and had then resumed her habit. She was not seen again. Over a year later a neighbor stated that both patients were still "fighting the bugs."

This is an illustration of the acquisition of the delusion in two persons simultaneously. It also demonstrates the inadvisability of informing such patients as these that an actual infestation is present in the hope that such agreement with the patient's belief, followed by therapy known to be efficient, will result in a "cure." Such a course only serves to give the patient a scientific confirmation of the presence of his infestation and fixes the delusion still more firmly. Too many physicians are satisfied to make the presumptive diagnosis of infestation with parasites on too little evidence; in cases such as these it causes untold harm.

Another instance in which both husband and wife presented the delusion has been observed; it is worthy of recording as an illustration of the extent to which the mental processes of such patients may take them in the study of their "parasites."

CASE 5.—The husband, 76 years old, presented a bizarre series of deep excoriations involving the face, nape, hands, anterior chest wall, forearms, and shins. The wife, 72 years old, presented similar lesions but to a less degree. A portion of their story, written by the husband in response to a request for details, is better presented unexpurgated.

"These notes are written to help in a measure to warn anyone who might be so unfortunate as to be so afflicted and urge them to take immediate steps to eradicate these terrible parasites.

"Subject, Fleas. The one we deal with in these few notes we believe to be the chigre (chigger) flea, commonly called the jigger, originally coming from the country of Peru or the West Indies.

"My wife first had what we called hives, which we treated accordingly. We did not know of the seriousness of the affliction until our bodies had been seeded to the limit; a low estimate would be 400 habitations. The source of the infection was rats coming into our unfinished service porch leaving us plenty of unbidden guests.

"The human flea buries itself under the skin and sets up its domicile and then proceeds to raise a family. Incubation period is five or six weeks. We know, for we have been through it several times.

"The forerunner of these fleas, the parent, is whitish, a quarter of an inch long and flutters around. On settling on the body it stands on end and drills its way into the skin until completely hidden. Then it lays its eggs. After incubation these sores erupt like volcanoes and pour out No. 2, which we term the gunpowder type. These are without locomotion, but they get there just the same. They are very sharp and settle into the skin immediately. Next is No. 3, the cucumber seed type. No. 4 is a round bug termed the rice flea and No. 5 is round like a tomato seed, both white and dark, one-eighth inch in diameter. No. 6 is round, needle-like and a quarter inch long. This one is ever present in large numbers. No. 7 goes unnamed, twice the width in length, found in great numbers and possibly the excrement of fleas. No. 8, what we are pleased

to call the emery type, feels like emery dust. It infiltrates the skin, leaving no trace or spot to mar the surface but makes itself known by a distressing irritation. Nothing to do but rub them out by a quick circular motion of the hand, then scraping with a knife blade will remove them. No. 9 is small but white in color and extremely plentiful. No. 10 is the 'mystery flea' equipped with wings and not very plentiful.

"What makes it so difficult to eliminate these parasites is a system of fortifications mostly under cover even to a depth of one-half inch."

After several more closely typed pages he further states, "We are not going into details but this parasite is the most loathsome thing we have met up with."

DIAGNOSIS

It is, of course, necessary to give the patient the benefit of the doubt until an adequate examination has been made and has failed to reveal evidence of actual parasitic infestation. I must admit that on one occasion a patient recited to me a story which seemed to indicate beyond any doubt that she was suffering from the delusion of being infested with head-lice obtained in a most bizarre manner. All smugness in my mental attitude was dispelled immediately, however, upon examining her nape; for it was evident that a heavy infestation with Pediculus capitis was actually present.

It is well to reiterate that such patients as those previously mentioned deserve a scientific approach to their problem until an accurate diagnosis has been made. It may happen that the "materials" brought in will actually prove to contain pathogenic organisms. Also the opportunity should not be missed of discovering an adequate reason for the existence of the symptom complained of; for example, the delusion may be superimposed on any pruritic disease, either cutaneous or systemic. In the majority of instances it will be easy to assemble sufficient facts to make the diagnosis incontrovertible.

SYMPTOMS AND SIGNS

The symptoms complained of by patients exhibiting the delusion of parasitosis cover the entire range of sensations of which the skin is capable. Pruritus, of course, predominates, but creeping, crawling, moving, biting, scratching, sticking, digging, burning, clicking, irritation, and simple aggravation have been described. Usually associated with these are a variety of symptoms referable to bodily organs other than the skin too extensive for recording here. Superimposed on all these is the component of severe mental illness which is always present.

From the wide variety of findings on examination of such patients heretofore observed, it seems likely that any variety of dermatosis can be expected in association with this delusion; it is also true that in some cases no abnormality of the skin whatever will be discernible. One case has been observed in which the delusion had recently been superimposed on psoriasis of many years' duration. It appears that there need not necessarily be any relationship at all between the delusion and the condition of the skin itself.

Aside from totally unrelated dermatological entities, the lesions in this disorder will usually be of one of three types: those injuries due to the patient's attempts to remove the "parasites" by excoriations with the fingernails or other instruments or methods; those due to chemicals, medicaments, and soap utilized to kill or remove the parasites, and manifestations of psychosomatic dermatoses. The first two do not lend themselves to discussion here; the last appears worthy of consideration.

RELATIONSHIP TO PSYCHOSOMATIC DERMATOLOGY

Of 16 patients suffering from this delusion personally observed, six presented lesions of the type usually referred to as neurodermatoses. There were three with lichen simplex chronicus of the nape; two of these also presented similar lesions on the neck and hands; all three were women. One patient presented the typical picture of disseminated dry neurodermatitis after he had been persuaded to avoid overmedication long enough to allow a severe dermatitis venenata to subside. Two instances of vesicular eruption of the palms and soles were seen. In all six cases the element of anxiety and apprehension seemed to be notably accentuated, whereas it was apparently totally absent in some of the patients exhibiting no neurodermatitic syndromes. In one of these cases the dermatosis disappeared while the patient was under observation in a manner which may be instructive.

Case 6.—A 49-year-old woman had been in the menopause for three years, during which time she had been becoming increasingly depressed about the future. She had spoken of suicide many times, was extremely irritable and nervous, slept poorly, cried a great deal, and moved continually about the home making futile efforts of short duration at doing the housekeeping. She presented large plaques of dry localized neurodermatitis, stated to be of two years' duration, involving the nape, sides of the neck, and the extensor aspect of one elbow. The plaques were said to be "full of parasites" which she could rub out with a coarse towel. They were described as tiny white threads which came out of the skin only at night. She brought to the office approximately 1 gm. of epithelial debris which she said represented bugs which she had killed the previous night by "rubbing them with the towel into little balls."

In spite of treatment by several physicians using various methods, including x-radiation and medications both internal and external, the plaques persisted. About a year later, however, they began to regress spontaneously and cleared entirely during the next four months. During this time it was noted that she was becoming less agitated and more depressed. Finally she was spending most of each day gazing off into space, expressing little or no concern about events occurring around her. She still maintained the delusion that parasites were present but stated that, since she had been so unsuccessful at eradicating them, she had simply managed to get used to them and was at last able to tolerate the itching. Psychiatric consultation resulted in a diagnosis of involutional melancholia with profound depression.

In this case it seems that as the anxiety disappeared so did the cutaneous lesions, although the delusion persisted. Conversations with psychiatrists have led me to believe that anxiety may be associated with any psychosis (as well as with any of the psychoneuroses). It may, on the contrary, be absent from examples of all such disorders (except, of course, from true anxiety neurosis) without affecting the profundity of the mental illness. If the patient ceases to fight against the disorder, the anxiety component may disappear. It may also do so if the patient finds an adequate psychologic shield against his troubles, even though this deepens the actual mental aberration.

It seems likely that whether or not in patients with mental illness neurodermatoses develop depends not so much on the type of mental change as on the degree of anxiety associated with it or engendered by it.

TREATMENT

The study of this series of 34 cases of the delusion of parasitosis has served only to strengthen the opinion previously expressed that the syndrome invariably indicates such a profound degree of mental illness as to be far beyond the scope of what is ordinarily termed "superficial psychotherapy." Once the presence of such a delusion has been incontrovertibly established, the dermatologist should thereafter

limit his efforts to therapeutic measures reasonably directed toward the amelioration of whatever actual disease of the skin many be present, carefully avoiding creating the inference in the patient's mind that the "parasitic" infestation is being treated. In addition, he should do his utmost to convince the patient that the disorder is psychologic in origin and that psychiatric care should be sought without delay.

Because of the paranoid nature of the illness the patient will usually lack sufficient insight to entertain even the slightest possibility that mental abnormality is the explanation; all such suggestions will be met with extreme antipathy, frequently with anger and scorn. Only 5 of the 34 patients in this series obtained psychiatric consultation; none were cured. Case 3, previously recorded here, is the only instance in which the delusion disappeared permanently; this was not the result of medical care but occurred because the circumstances causing the psychoneurosis were removed.

Involutional melancholia can be helped greatly by electroshock therapy; dementia praecox perhaps by insulin shock. Rarely will psychoanalysis serve a useful purpose. A delusion such as this develops as a protective mechanism for the mind against the onslaught of mental turmoil of large degree. Nielsen and Thompson [2] have likened it to the attempts of the physical body to isolate and wall-off an inflammatory process when an assault is made upon the tissues by a noxious agent. An abscess which has been successfully localized and surrounded by a protective wall may cause extensive damage if the wall is broken; in the same manner the effect might be devastating and result in dementia if the mentally ill person were to have complete recognition of the extent to which his delusional systems have gone unless an adequate alternative protective mechanism is immediately substituted. The psychoanalyst is likely to probe just far enough to become aware of the situation and then state that the patient has made a better adjustment to the turmoil than any he could offer as a substitute.

SUMMARY

Sixteen cases of the delusion of parasitosis observed by me and 18 cases in which the details were related to me have been studied.

Without more intensive psychiatric study than is usually sought or obtained by such patients, accurate psychiatric classification can seldom be made.

While this delusion apparently usually occurs in a truly psychotic individual, it may be found occasionally as a manifestation of a severe psychoneurosis. It is always, however, an indication of mental aberration beyond the scope of treatment by practitioners other than those well trained in psychiatry.

The delusion probably frequently affords to the patient a better mechanism for adjustment than any substitute which psychiatry could offer.

The dermatologic manifestations accompanying this disorder may be totally unrelated to it. They may be the result of physical or chemical trauma inflicted by the patient upon himself. The remainder are classifiable into the neurodermatoses, in which case anxiety seems to be the causative factor.

3875 Wilshire Blvd.

2. Nielsen, J. M., and Thompson, G. N.: Engrammes of Psychiatry, Springfield, Ill., Charles C Thomas, Publisher, 1947, p. 111.

THE MANHATTAN DERMATOLOGICAL SOCIETY

Its First Fifty Years (1900-1950)

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THE MANHATTAN Dermatological Society is in many ways a unique organization. It is the second oldest dermatological society in New York, giving priority to the New York Dermatological Society which, founded in 1869, is the oldest in the world. There was never any real rivalry between the two Societies, and they were conducted on much the same plane. As a matter of fact, of the 55 active members elected to the Manhattan Dermatological Society within this 50 year period, 14 have also been members of the New York Dermatological Society. What the Manhattan Dermatological Society has meant to its members during the 50 years of its existence is evidenced by the fact that there have been only eight resignations during that time.

For many years the Manhattan Dermatological Society was probably the most difficult medical society in the world to enter. If a member desired to propose Dr. Doe, he did not do so from the floor, but advised the Executive Committee of his intention, whereupon the secretary wrote each active member, asking if he had any objection to such action. If there was a single objection, and reasons did not have to be given, the name of Dr. Doe was promptly dropped by the Executive Committee. If no objections were received, a unanimous vote of the members present was still required. Since 1934, elections have been more orthodox, and a majority ballot is sufficient. However, the Society is still small and intimate, numbering 18 active members, and restricts its scientific work (as does the New York Dermatological Society) to the presentation of unusual dermatoses, with all necessary laboratory data.

The Manhattan Dermatological Society was organized on Nov. 21, 1900, at the home of Dr. William S. Gottheil, 144 W. 48th St., and the men responsible for its inauguration were Drs. Gottheil, Ludwig Weiss, Edmund L. Cocks, J. P. Oberndorfer, Robert Abrahams, and M. W. Franklin.

At the second meeting held on Jan. 4, 1901, at the residence of Dr. Ludwig Weiss, Drs. Asha Bleiman and Jacob Sobel were present as new members. Dr. Gottheil served as president of the Society and Dr. Sobel as secretary. This first meeting was highly successful despite the presence of only eight men. Six interesting cases were presented. At the executive session Dr. Weiss suggested that

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children be presented first, a rule adopted and still followed 50 years later by all the dermatological societies in New York. Dr. Weiss also suggested that members read a history of each patient presented, with the idea "that our maiden effort be of high order." Dr. Abrahams suggested that means be devised for the publication of the scientific transactions of the Society. All these transactions, from the first scientific meeting on Jan. 4, 1901, to October, 1906, were published in the *Medical Record* and *Medical News*, and from 1906 to date by the (A. M. A.) ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY.

In my opinion, the presentation of six unusual dermatoses by 80% of the members present and the proposals cited above at a first executive session are ample evidence that the type of men who founded the Manhattan Dermatological Society were of the highest order.

Like many scientific undertakings which prove successful from the beginning, a lever in the form of a man with great driving force is needed; much of the early success of the Society was due to the tireless energy and devotion of William S. Gottheil. Ludwig Weiss most ably assisted him and was equally devoted to the Manhattan Dermatological Society. The first yearly report, dated Jan. 3, 1902, recorded that nine regular meetings had been held and 84 cases presented.

Dr. Gottheil was born in Berlin on Feb. 5, 1859. At the age of 11 he came to America with his parents after spending several years at a school in Manchester, England. He received his A.B. degree from Cornell University and his M.D. degree from the College of Physicians and Surgeons, Columbia University. He served his internship at the old City Hospital from 1881 to 1883 and developed a deep interest in that institution. He became attending dermatologist in 1901, remaining there until his death in 1920. It was here that he made most of his *moulages* and at the time of his death he had a large collection of them. His teaching career started at the Post-Graduate Medical School and Hospital as adjunct professor of dermatology from 1907 to 1912, when he resigned to become professor of dermatology at Fordham University—the chair he held until his untimely death. He did much original work and was greatly responsible for the use of the insoluble mercurials, particularly the salicylates, solid carbon dioxide, and autoserum. This professorship at Fordham is a splendid example of tolerance. Dr. Gottheil's father was a well-known rabbi, yet the son was offered a professorship in a Catholic university.

Dr. Gottheil died dramatically. He passed from health to death in a few minutes. Dr. Ochs, an old friend, who was his assistant at Fordham, told me that, on returning from the college with Gottheil on a Jewish holiday, Gottheil asked him to stop at his house and sample some excellent wine he had just bought. Ochs, who had had a thigh operation near the hip, was on crutches, and while Gottheil was uncorking the bottle, laughing in the meantime at some joke, Ochs was having difficulty in removing his overcoat. Suddenly he heard a crash back of him and, turning, saw Gottheil lying on the floor dead. He had had no premonitory symptoms whatsoever.

I feel that Ludwig Weiss (1852-1930) deserves as much credit as Gottheil as the founder of the Society. He was born on Feb. 7, 1852, at Eszbergom, Hungary, and was graduated in medicine in Vienna in 1877. Shortly thereafter

he began the practice of medicine in New York. For a short time he lectured on skin diseases at the New York Post-Graduate Medical School and Hospital (1893 to 1895). Although Dr. Weiss served on the staffs of several hospitals, his greatest devotion was given to the German Polyclinic, of which he was president from 1900 to 1905, serving as chief of its dermatological department from 1883 to 1930. Dr. Weiss was the author of many worth-while articles and also translated several books from German into English and from English into German. Weiss was deeply interested in the endocrine glands and their relation to various dermatoses and wrote much on the subject, as well as on tuberculin therapy and roentgen therapy in dermatology. He was an honorary member of the *Deutsche medizinische Gesellschaft*. At a memorial service held at the Academy of Medicine on March 16, 1930, under the auspices of the German Polyclinic, Rabbi Stephen S. Wise said in part,

> He was a friend of men, a man who took his calling seriously and gave to it all that was in him—his best powers, his entire strength, his utter consecration. He was a true physician. As such he ministered to men, as such he will be remembered and his name long be blessed.

Edmund L. Cocks (1856-1911) served in Dr. Bulkeley's Division at the New York Skin and Cancer Hospital from 1902 to 1910. He preceded Dr. Gottheil as professor of dermatology at Fordham University, and also served as chief of the dermatological clinic both there and at Harlem Hospital. He was president of the Harlem Medical Association. His excessive modesty masked a deep clinical knowledge of dermatology, and he was devoted to the Manhattan Dermatological Society. Dr. Gottheil at the end of Dr. Cocks's obituary, states: "To the end of his life he remained an earnest learner and worker in his chosen field, and in his demise the specialty loses a quiet but most useful follower."

Dr. Benjamin F. Ochs was present as an active member at the third regular meeting of the Society on Feb. 1, 1901, and during the 32 years that he remained an active member he rarely missed a meeting and presented many interesting cases. He was an able discussant and first introduced the use of potassium permanganate solutions in the treatment of tinea of the feet. Dr. Ochs died in June, 1933.

Dr. Jacob Sobel, the first secretary of our Society, is still living and in active practice, and we are greatly indebted to him for the careful recording of the minutes of the Manhattan Dermatological Society. He resigned from the Society in October, 1905. He was interested in dermatology but did not make a specialty of it. A graduate of the College of Physicians and Surgeons in 1895, he has practiced medicine for 55 years. Among the best known of the older members of the Society who have passed into the Great Beyond, the names of Mihran B. Parounagian, Ludwig Oulmann, David L. Satenstein, Jerome Kingsbury, Charles W. McMurtry, J. Frank Fraser, Fred Wise, and our first honorary member, George H. Fox, claim attention. Among the more recently elected members who have died are E. William Abramowitz and Girsch D. Astrachan.

Dr. Mihran B. Parounagian's devotion to the Manhattan Dermatological Society was phenomenal; from the time of his election in 1905 to his death on June 13, 1946, he rarely missed a meeting. He was its secretary from 1907 to 1912 and again from 1931 to 1937. He served as President in 1912 and 1929 and was a

member of innumerable committees. In his 41 years as a member he must have shown hundreds of patients before the Society. This was easy, as he was the head of the departments of dermatology and syphilology at the Gouverneur, City, and Bellevue Hospitals, each members of the hospital system of the City of New York. He taught his specialty at different times at the New York Post-Graduate Medical School and Hospital, the University of Vermont, and the School of Medicine of New York University. His outstanding work was done in syphilology and its therapy. The attendance at his syphilis clinic at Bellevue Hospital more than tripled during his incumbency. The observation of the hundreds of syphiloderms presented by him for 30 years before the Society convinced me of his uncanny ability in clinical diagnosis.

Dr. Ludwig Oulmann (1875-1940) was born in Stuttgart, Germany, on June 2, 1875. He was graduated in medicine at Würzburg and did postgraduate work at Nürnberg and Berlin. He served with the Imperial German forces in China during the Boxer Rebellion. As a young physician he was a ship's surgeon for the Hamburg-American Line and saw much of the world. He worked with Röntgen and enjoyed a close friendship with Unna. He began practice in New York in 1904, but almost every year he visited his beloved Germany. He was elected to the Manhattan Dermatological Society in January, 1906, and remained a member until his death. He was fond of the Society, regular in his attendance, and frequently took part in the discussion of cases, besides presenting many of his own. Oulmann joined the dermatologic staff of the Lenox Hill Hospital early in his career and served it until his death.

Dr. David L. Satenstein (1880-1943) was born in New York on Feb. 22, 1880, and received his medical degree from the College of Physicians and Surgeons in 1902. He spent two years in Europe in intensive postgraduate study. Shortly after his return to New York he became associated with Dr. Gottheil at the old City Hospital, and spent much time in assisting Dr. Gottheil in making *moulages* of various dermatoses. Possibly the minute care required to make good ones, besides his own inclinations, started him on one of his two lifetime devotions, histopathology. The other was the Manhattan Dermatological Society. Dr. Satenstein was elected to the Society in April, 1907. He served as secretary from 1912 to 1930, probably the longest tenure of this particular office in any dermatologic society. He entered his life's work when he became affiliated with the dermatologic service of the Vanderbilt Clinic under Fordyce in 1915 and remained there until shortly after Fordyce's death in 1925.

Upon his resignation he joined Dr. George M. MacKee at the New York Post-Graduate Medical School and Hospital, where he became associate professor and chief of its department of histopathology of the skin. He continued this work after 1934, when the New York Skin and Cancer Hospital was taken over by the Post-Graduate Hospital. He kept this up until his death, so that for 25 years or more he devoted most of his time to the microscopic interpretation of diseases of the skin. He was a painstaking teacher, and often spent hours over several boxes of slides, choosing only a few which presented the most characteristic features he was seeking to teach. Younger dermatologists remember him gratefully for their start in dermatopathology, an invaluable aid in the study of skin diseases as a whole.

Dr. Jerome Kingsbury (1871-1944) was elected a member of the Manhattan Dermatological Society in May, 1908. This was the first dermatologic honor he received and, in evidence that it was not misplaced, six months later he was elected to the New York Dermatological Society and five years later to the American Dermatological Association. Kingsbury was graduated in medicine from Bellevue Hospital Medical College in 1897 and interned at the old New York Skin and Cancer Hospital from 1898 to 1900. He then began a long and faithful service on its dermatological staff until 1920, when he resigned. In 1929 he accepted a reappointment as attending physician and remained until the amalgamation of the New York Skin and Cancer Hospital and the New York Post-Graduate Medical School and Hospital on Sept. 1, 1934, when he again resigned. Dr. Kingsbury was visiting dermatologist at the City Hospital from 1920 to 1936, where he became expert in the diagnosis of leprosy. At various times he had charge of the dermatologic services of the Presbyterian Hospital Dispensary, Midtown Hospital, and Northern Dispensary, of which he was a trustee. He served as one of the professors of dermatology and syphilology at the New York Polyclinic Medical School and Hospital from 1925 until his death. Dr. Kingsbury wrote the text of "Portfolio of Dermachromes," published by Rebman, New York, in 1913, containing the colored plates which appeared in the fourth edition of Jacobi's "Atlas." The text was entirely new and clearly presented the American viewpoint of dermatology at that time.

Dr. Charles W. McMurtry (1872-1914) was born in Pittsburgh on May 1, 1872, and was graduated in medicine from Harvard University in 1897. He immediately went to Europe to study dermatology and syphilology, where he remained for 10 years. Three of the 10 were spent in Breslau under Professor Neisser, where McMurtry held the position of first assistant in dermatology in the Kaiser's Clinic. For a number of years he studied in Berlin under Professor Lesser and in Paris under Fournier and Darier. Under the last he perfected his knowledge of the gross and microscopic pathology of diseases of the skin. He returned to New York in 1908 and served the New York Skin and Cancer Hospital as clinical assistant on Dr. George Henry Fox's service from 1909 to 1912. He resigned at the same time as Dr. Fox. He became an instructor in dermatology at the College of Physicians and Surgeons in 1910. He donated a large and fully equipped pathological laboratory to the dermatological department of the Vanderbilt Clinic and was an avid worker in histopathology. Dr. McMurtry was elected to the Manhattan Dermatological Society in February, 1913, and regularly attended its meetings until his premature death about one and a half years later.

Dr. J. Frank Fraser (1867-1943) was elected a member of the Society in April, 1928. He was born at West River, Nova Scotia, on Jan. 8, 1867, and received his medical degree from Bellevue Hospital Medical College in 1892. He first devoted much time to the study of pathology in the laboratory of his brother, Alexander Fraser, a professor of pathology at the School of Medicine of New York University. Fraser later specialized in dermatology and became one of the leading histopathologists in New York. He held many important positions in New York, among

which were chief of clinic of the Department of Dermatology and Syphilology at Cornell University Medical College, and associate visiting dermatologist at the New York Hospital. In 1935 he was made chief of clinic at the New York Skin and Cancer Unit of the Post-Graduate Medical School and Hospital. He was a splendid teacher and had many occasions to demonstrate this fact as associate professor in dermatology at Cornell University Medical College and the Post-Graduate Medical School and Hospital. Dr. Fraser wrote a number of interesting articles on mycosis fungoides, presenting a number of new ideas on its pathology. In fact, the majority of his medical articles presented a new viewpoint on the subjects. In discussion, Fraser was modest but not reticent and confined his remarks to pertinent facts. It was, therefore, a great pleasure to listen to him.

Dr. Fred Wise (1882-1950) was elected a member of the Manhattan Dermatological Society in February, 1910. He was graduated from the College of Physicians and Surgeons in 1904 and interned at the New York Skin and Cancer Hospital from 1904 to 1905. This, apparently, made him decide to take up dermatology as his life's work. His first position at the above hospital in 1906 was as clinical assistant on Dr. Torek's service in the cancer department. He served there until 1910 when he became a clinical assistant in dermatology on the service of Dr. George Henry Fox. When Dr. Fox resigned, in 1911, most of his staff resigned with him, including Dr. Wise. Shortly thereafter Dr. Wise entered his life's work by his early association with Dr. John A. Fordyce, who was professor of dermatology at the College of Physicians and Surgeons. Dr. George M. MacKee, Dr. Fordyce's chief of clinic at Vanderbilt Clinic, and an instructor in dermatology at the College of Physicians and Surgeons, soon recognized Dr. Wise's superlative abilities, and they became close friends. At about this time a famous foursome joined Fordyce's staff, Drs. Isadore Rosen, David L. Satenstein, Max Scheer, and E. William Abramowitz. For nearly 40 years these four were associated with Wise and MacKee; and all six ascended the ladder of fame together, reminding one of Alexandre Dumas' "Three Musketeers" and his famous "one for all and all for one." Despite much reading and writing on the historical side of medicine, I do not recollect ever coming across a similar instance. As MacKee was promoted, Wise kept step with him, and on his own wrote so many articles casting new light on dermatology that this alone brings him fame. His teaching both at the Vanderbilt Clinic and later at the Post-Graduate Hospital helped make better dermatologists in the ranks of the aspirants to our specialty.

Dr. E. William Abramowitz (1886-1949) was elected to the Society in 1937. He received a degree in medicine from the University and Bellevue Medical College in 1907 and in 1914 joined the dermatological department of the Vanderbilt Clinic, Columbia University, where he joined the famous group of Rosen, Satenstein, Scheer, Wise and MacKee, which were to remain together for so many years in teaching and clinical work at both the Vanderbilt Clinic and the New York Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital. Dr. Abramowitz was attending dermatologist and syphilologist to many hospitals, the principal ones of which being University, Gouverneur, and Jewish Memorial Hospital. He was Clinical Professor of Dermatology at the

New York Post-Graduate Medical School and Hospital from 1927 to his death in 1949. Dr. Abramowitz wrote many important articles, but he will be best remembered for his original work on drug eruptions and his introduction of 40% sulfur ointments in the treatment of seborrheas.

Dr. Girsch D. Astrachan (1892-1946) was elected to the Society in 1944. He was born in Russia, served from 1915 to 1917 as a captain in the Russian Army in World War I. He was a prisoner of war in Germany from 1917 to 1920. He was graduated in medicine at Jena, Germany, in 1923, and came to New York in 1924, where he became an intern at the Metropolitan Hospital (1924-1925). He served on the dermatologic staff of the Flower-Fifth Avenue and Metropolitan Hospitals, but his most important connection was attending dermatologist and syphilologist to the New York Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital. He had written a number of medical articles, and his future seemed bright when he died prematurely on April 30, 1946.

We have paid our respects to the illustrious past of the Manhattan Dermatological Society, and we will now relate what it has accomplished through its present members, who form but a small group. Currently, five members of this Society are professors or associate professors at the College of Physicians and Surgeons, Columbia University, the School of Medicine of New York University, and Cornell Medical College. There is hardly an important dermatological department in many of the leading hospitals in New York which has not had a member of the Society on its staff at some time or other. All of the dermatological societies, national and local, have had at least one chairman who was a member of this Society. Four of our members became president of the American Dermatological Association. Dates and the recital of achievements in written history are as cold as frozen pudding and almost as uninteresting, but they are highly essential for future reference, so it might be well to give some of my impressions gleaned from a membership in the Manhattan Dermatological Society since 1913.

Gottheil was a handsome man with clear, piercing eyes, and a closely cropped mustache and Van Dyke beard. His hair was thick and gray, as were his mustache and beard. His discussions were pertinent and confined to the patient presented. He avoided the common digression, occurring even today, of relating the symptomatology of a patient with a similar clinical picture to the one presented. He was neither self-conscious nor anxious to make an impression, nor was he patronizing to a young tyro like myself, but was, on the contrary, affable and jovial. I observed him laugh uproariously at jokes, or at mishaps in presenting a patient. He never missed a meeting, and his devotion to the Society was plainly evident. He enjoyed the four- or five-course suppers served after the meetings at about 11 p.m. or 12 m. and lasting until 1 or 2 a. m. Thes meals were complemented with wine, beer, and cordials. At that time, cigars were more popular than cigarettes, and nonsmokers were rare; hence, the atmosphere of the dining room soon resembled a fog of London proportions. No one minded, for most of us had a corona-shaped cigar in our hands. These suppers were an institution, and, in those days, as most of the members lived in private houses or large apartments, a large dining room was as common as the kitchen. There was no rivalry as to their size, nor was there any attempt at sumptuousness, but these suppers were always large enough to cause a great

dislike for food for the ensuing day or two. The most popular ones were those served at the Parounagian and Weiss homes—Armenian cooking at Parry's and Hungarian at Weiss's. Both men were charming hosts and plied us with the most tempting dishes of their respective countries. They were overjoyed when their turns came to entertain and were displeased if we left before 2 a.m. What struck me most forcibly was the ease with which these suppers were consumed without untoward effects. At one special supper at Parounagian's when I happened to sit between Weiss and George Henry Fox I was awed to see them consume several different courses of meat plus soup, entrees, salads, and desserts, in complete comfort and evident enjoyment. Fox was then around 78 and Weiss about 70, while I, many years younger, felt like a balloon. How I did enjoy it nevertheless! George H. Fox's stories, his witty speeches, the badinage, sociability, and camaraderie were all delightful. Life seemed less tense in those days, less hurried.

Dr. Weiss, born in Hungary, educated in Vienna, and retaining all the qualities of an inhabitant of *alt Wien* in the days of the younger Johann Strauss, was thoroughly American. His English had no trace of a Teutonic accent, and he completely lacked the authoritative attitude so common to many Austro-Germans of that period.

I first met Dr. Edmund L. Cocks in 1909, when I worked under his direction on Dr. Bulkley's service at the old New York Skin and Cancer Hospital, and at the same time served as Dr. Bulkley's office assistant. Dr. Cocks, who had preceded me at the clinic by seven years, had been promoted to the new-case room, where he inscribed in a large ledger the name, age, address, history, diagnosis, and treatment in each new case. After a week or two of observation I inherited the book and the pen. This gained me the advantage of observing every new case entering Dr. Bulkley's service, besides having the added advantage of careful instruction by Dr. Cocks, as well as by Dr. Whitehouse, the chief of clinic. Cocks's diffidence masked an extraordinary degree of diagnostic acumen. I shall remain eternally grateful to him for the great pains he took in directing my first dermatologic steps.

Dr. Benjamin F. Ochs had a fine personality. How deeply he loved dermatology was impressed upon me when after observing 20 or more cases and discussing them *ad infinitum*, followed by supper lasting until about 1 a.m., we would leave together for our homes in uptown Manhattan, and Ben would not let me go home before explaining to me, on some street corner, the intricacies of an atypical form of a dermatosis he had observed at the hospital that day. Ochs's high amputation of one thigh caused him to use crutches all his life. He told me that on an unusually hot day in mid-August he was waiting in the sun for a street car. Feeling the perspiration streaming down his face, he removed his hat and held it bottom-side up against his crutch, and, while wiping his brow with his other hand, he felt something fall in his hat. To his surprise, it was a quarter, dropped by a passing lady who mistook him for a beggar. He kept it for many years as a lucky piece. The mistake was doubly ludicrous because Ochs was a meticulous dresser and was always well groomed. On one of the many occasions that I served as host to the Society, I received from Ochs the usual postcard answer in the affirmative. Under his name he had written "Count me in the crowd, but do not crowd me out." It is in my collection.

Dr. Ludwig Oulmann, unlike Weiss, remained a foreigner despite residing in New York for 36 years and did not become an American citizen until after the Armistice. Oulmann was a member of many German societies. Even his professional connections were German. It is therefore not surprising that throughout World War I his sympathies were with his fatherland. In consequence things were difficult for him in the Manhattan Dermatological Society, but he and the Society weathered the storm, perhaps because Oulmann was companionable, pleasant in his ways, and an excellent dermatologist. He had been a student under the leading men of Germany's golden age of dermatology. My relations with him were always agreeable, and he must have thought something of me, for without solicitation he gave me several autographed letters written to him by his former teachers. I last saw Oulmann's earthly remains under peculiar circumstances. After his death, I went to pay my respects to him at his apartment on upper Madison Avenue, the site of many good suppers in German style when he was host for The Manhattan Dermatological Society and plied us with *Willkommen* glasses of German imported beer. Oulmann lived in a very large, old-fashioned apartment on the ground floor, and, except for a housekeeper, he had lived alone. I was admitted by this housekeeper and went to the sitting room where the body lay. No one was there, and everything was as silent as the tomb he was soon to enter. As I passed the threshold, I noticed something moving on Oulmann's chest and I immediately recognized his old mongrel dog to which he was greatly devoted. The dog, in trying to prevent me from approaching the coffin, was running all over Oulmann's body and barking furiously, whereupon the housekeeper came to take him away. She told me that the dog persisted in jumping into the coffin and laying his head on Oulmann's face or neck, had refused all nourishment, and spent his time walking and whimpering through the empty rooms. I did not leave Oulmann's apartment for the last time with dry eyes.

One cannot talk of the personal side of Dave Satenstein without Parounagian, as both men were the Manhattan Dermatological Society. They were more or less perpetual secretaries and, of course, served as presidents, but Dave put one over on Parry by serving as both president and secretary of the Society at the same time for one year, a record that I believe has never been rivaled in any dermatological society at any time or place. They served on innumerable committees. In a single instance I happened to also serve on a committee with them; what earthshaking thing we were considering does not matter, but what does is that we met after a monthly meeting of the Society, and Dave drove us to Parry's home. By that time it was about 1:30 a.m. Parounagian would not let us attend to the business part of our meeting until Mrs. Parounagian had brought a tray of eatables and choice wines; even then, he was not satisfied with the wines and left the room to get a bottle he had saved for some particular occasion. The business was dispatched in a short time, and the conversation turned to dermatology and medical politics. When I left at 3 a.m., Dave and Mihran were still at it. I mean the talk fest, not the bottle. Lest my occasional mention of the "cup that cheers" in this recital of the social side of the Manhattan Dermatological Society in its early days might lead my readers to think that its members imbibed freely, I want it understood here and now that the display of the choicest of "fire water" was always used sparingly and at

the 240-odd dinners, collations, and suppers of the Society that I have attended since 1913, no one showed the slightest indication of having had too much. Dr. Satenstein devoted all his energies to the progress of the Manhattan Dermatological Society and his activities were terminated only when his long cardiac illness kept him first at home and then in various hospitals.

Dr. Jerome Kingsbury had an extraordinary degree of reticence, which was manifested professionally but not socially. He was a delightful companion at dinner. His modesty was such that he neither spoke of himself nor his work, and it was difficult to induce him to discuss the cases presented. This modesty is clearly demonstrated by the following incident. Two of his colleagues were attending a moving picture, when, on the screen, to their surprise they saw Jerome Kingsbury entering a balloon to take part in an international contest. One of them later said to him, "Why didn't you tell us you were a balloonist?," to which Kingsbury answered, "Why, you never asked me." For many years he had been a licensed balloon pilot, a charter member of the American Institute of Aeronautical Engineers, chairman of the Balloon Committee of the Aero Club of America, a member of the Early Birds, and a competitor in many national and international balloon races; yet he was so modest that, despite frequent association, he never spoke to his friends of his exploits in these fields. He was also a life member of the United States Revolver Association, and the tops of his bookcases in his office were filled with cups he won in tournaments by his expert marksmanship. I was with him at the old Skin and Cancer Hospital, at the City Hospital, and Presbyterian Hospital two or three times a week for some 25 years, and, though we worked on the most friendly terms possible, his reserve was such that I did not even know where he resided. Yet, I was very fond of him and I learned to appreciate his expertness in sight diagnosis. Kingsbury was deeply interested in military affairs and was a member of the famous Seventh Regiment of New York. He served as a captain in the Medical Corps in France throughout World War I, not as a dermatologist, but as an operating surgeon in the field. After the war he held the rank of colonel in the Medical Reserve Corps.

Dr. Charles W. McMurtry had a delightful personality and, despite his father's great wealth, worked harder in dermatology than if he had to make a living in it. His father, a Scotsman, was chairman of the Board of Directors of the American Sheet and Tin Plate Co. (a subsidiary of the United States Steel Co.), a long-time associate of Andrew Carnegie, and a millionaire in his own right. McMurtry, born in Pittsburgh, had no inclination to follow his father in the steel industry. Early in life he became aware of his inclination for science, which he first gratified at the Massachusetts Institute of Technology, and later in the medical department of Harvard University. He then spent 10 years in the intensive study of pathology and dermatology in Germany and France, thereby receiving longer training in dermatology and histology than anyone I know. He donated a large and fully equipped pathology laboratory to the Dermatology Department of the Vanderbilt Clinic and worked there until his death.

McMurtry was fond of the Manhattan Dermatological Society and regularly attended meetings. He discussed cases in a low tone and was far from well. On the occasions when we were alone together, he liked to converse in French. We

shared a common ailment, hyperchlorhydria, and we often spent an hour in a restaurant drinking a pint of milk apiece, as it temporarily relieved his hyperacidity after the Manhattan dinners. I went with him because I was fond of him and learned through him some fascinating points on the medical and lay history of Europe and its art. It is an odd coincidence that McMurtry died from general peritonitis following the rupture of an inflamed appendix. I have often wondered whether the hyperacidity had been caused by chronic appendicitis.

Dr. Frank Fraser was one of our most popular members. In his discussions he was serious, but not dogmatic. He spoke clearly and presented cases with complete pathological reports. As both histopathologist and clinician, he made many valuable contributions to dermatology, and I believe I can say without danger of contradiction he was a genius as a story teller. To see him rise at dinner, clear his throat, look around at his audience, and in his deep bass voice and Scottish accent, start his best-liked story, "The Strawberry and the Poop Deck," with all the histrionics of an actor of the old school, was an event to remember.

It seems strange to write of Fred Wise in the past tense. After 40 years of friendship, seeing him at every dermatological meeting throughout the country, listening to his valuable discussions, reading his original papers, presenting new facts in dermatology, and having him as a dinner companion, I believe he was liked so much by all of us largely because of his modesty and fear of hurting our feelings if he disagreed with our diagnosis. For dermatology, Fred had what the French call *le feu sacre*. He read most of the dermatological articles in the literature of both Europe and America, and, what is more, he remembered what he read. He did a wonderful job as the editor of the "Year Book." He was devoted to the Manhattan Dermatological Society, enjoyed our meetings, and never failed to be present at supper or collation.

Our first honorary member, Dr. George Henry Fox, was elected in 1913, and to our great joy refused to act as one, but came frequently to the meetings and suppers and regaled us with his stories and reminiscences. In his autobiography he states,

> In 1913 I was invited to become the first honorary member of the Manhattan Dermatological Society, which invitation I accepted with pleasure. I have since attended the meetings of the Society with varying regularity and enjoyed seeing the large number of rare and striking cases of skin disease presented by its active members.

Two of our honorary members, Dr. George M. MacKee and Dr. Howard Fox, were former active members for at least 30 years. They were regular in their attendance and very fond of the Society. Devoted to dermatology and working far "beyond the call of duty" because they loved it, they were given in return most of the dermatologic honors that could be given them. A detailed biography of each man can be found in the special numbers of the ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY issued in their honor, The one on Dr. Fox, by myself, and Dr. MacKee's, by Dr. Fox. Dr. Pusey's tribute in a special number in the ARCHIVES when he was still very much alive started the fashion, in my opinion an excellent one, of not waiting until a physician's death to write appreciatively about him, and eulogy is easier and more pleasant to read before death, than after. It may well be that in time the taboo against writing a man's life while he is still alive will vanish.

Active members of the Manhattan Dermatological Society in the order of their election, are as follows:

1913 Paul E. Bechet
1915 Isadore Rosen
1922 Max Scheer
1922 George C. Andrews
1935 Anthony C. Cipollaro
1937 George M. Lewis
1937 David Bloom
1937 Maurice J. Costello
1938 Wilbert Sachs
1940 Herman Sharlit
1940 Samuel M. Peck
1942 Jack Wolf
1944 Thomas N. Graham
1945 Frank C. Combes
1945 Nathan Sobel
1949 Orlando Canizares
1949 Paul Gross
1950 Perry M. Sachs

EPILOGUE

I hope that this history of the Manhattan Dermatological Society will achieve the purpose in mind, namely, to impress the reader with the importance of the role it has played in the progress of American dermatology. It has been affectionately nurtured by its members, and in turn the Society has added much to their dermatologic knowledge. Its past has been marked by continuous progress and its present gives great promise for the future. If its future members will watch over it with the same tender care as its founders, I shall have no cause to worry.

SOME OBSERVATIONS ON THE PATHOGENESIS OF PSORIASIS

OSKAR GANS, M.D.
FRANKFURT am MAIN, GERMANY

ON TAKING stock after many years, psoriasis remains a riddle, and little more is known today than was known in 1913, when Schamberg first reported on it to this society. A postulation for conditions which might lead to a psoriatic eruption would appear to be (1) a potentially psoriatic person and (2) an endogenous or exogenous impulse that causes this potentiality to become manifest. This statement implies that psoriasis is not an entity, i. e., not dependent on one single cause. Psoriasis appears to be one of those skin eruptions which develop because a particular skin can respond to many different injuries with the same morphological reaction. A rule long disputed seems to be proved again: The same cause can produce quite different morphological reactions on the skin, and quite different causes may produce the same morphological reaction on the skin.

This extraordinary, but nowadays generally accepted, fact helps us—as I hope to show—to understand the condition we call psoriasis. In a given potentially psoriatic person this constitutional anomaly may be dormant for the whole life, or it may from time to time become manifest because the "balance" in the body fabric is disturbed to a point where the proverbial "last straw breaks the camel's back."

LABORATORY STUDIES

I wish first to review briefly the facts as far as research has established them during the last 40 years. I shall discuss the results of work carried out in the laboratory of my hospital by myself and by my co-workers, Dr. Leonhardi, Dr. Krause, and Dr. Steigleder, during the last year.

The results of various studies are very contradictory, whether established by the simplest or the most complex methods.

Studies on psoriasis have developed the concept that the disease is on an infectious basis, as supported in the past by Unna,[1] Lennhoff,[2] Nobl,[3] and Schamberg.[4] This concept has lost much credence, as evidenced by articles by Lindenberg,

Read at the Seventy-First Annual Meeting of the American Dermatological Association, Hot Springs, Va., May 24, 1951.

1. Unna, P. G.: Neuere Erfahrungen und Anschauungen über Psoriasis, Med. Klin. **2**:1037, 1906.

2. Lennhoff, C.: Inokulationsversuche bei Psoriasis, Berl. klin. Wchnschr. **57**:782, 1920.

3. Nobl, G.: (*a*) Zur Kenntnis der Psoriasis arthropathica, Arch. Dermat. u. Syph. **123**:632, 1916; (*b*) Psoriasis, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, J. Springer, 1928, Vol. 7, Part 1.

4. Schamberg, J. F.; Kolmer, J. A.; Ringer, A. I., and Raizies, G. W.: Research Studies in Psoriasis, J. Cutan. Dis. **31**:698, 1913.

Werth,[5] Gandola,[6] Kierland,[7] and others. The leucocytes in the Monro abscesses have been considered as proof of exogenous infection but such sterile abscesses can be found in other noninfectious dermatoses, such as mercury dermatitis. Later, Civatte [8] stated that there are usually more monocytes than leucocytes in Monro abscesses. The so-called dermatotropic virus of Lipschütz [9] and Kyrle [10] has proved to be nonspecific in recent studies of the nuclei of the cells.[11]

Warzecha found positive Wassermann reactions in psoriasis to be nonspecific because of the lability of the albumin and globulin fractions in the serum. In the blood and spinal fluid of about 100 psoriatic patients he never found a genuine positive reaction. On the other hand, an outbreak of a psoriatic rash following acute febrile infectious diseases, angina follicularis, and polyarthritis is well known.

CLINICAL OBSERVATIONS

Hunger as well as overfeeding have been considered as causes of psoriasis. My own observations after the second World War and those of others during the first World War disprove these opinions [12] (Fig. 1).

Effects of Adrenal Extracts.[13]—Lasting effects of cortisone and corticotropin (ACTH) on psoriasis have not been seen in my clinic nor by Reiss,[14] or by Riehl [15] in Vienna. It is now well known that cortisone and corticotropin produce profound metabolic changes in normal persons, but so does fever or any acute infection. They all have a considerable, if temporary, effect on the "collagen" diseases.

The term "adaptation diseases," as far as psoriasis is concerned, appears to be just another name. Its use shifts the question to a different angle but does not solve the problem. The temporary effects of cortisone and corticotropin are, therefore, to be considered only as another factor, one of the many others dealt with above.

The same holds true for those changes in the general metabolism of the psoriatic patient which have been used to explain the abnormal parakeratotic reaction

5. Werth, J.: Ist die Psoriasis eine Wiruskrankheit? Dermat. Wchnschr. **115**:781, 1942.

6. Gandola, M.: Ricerche sull' etiologia della psoriasi e del lupus eritematoso in rapporto alle indagini di Lindenberg, Dermosifilografo **15**:591, 1940.

7. Kierland, R. R.: Attempts to Prove the Specificity of Streptococci Isolated from the Nasopharynx of Patients with Psoriasis, J. Invest. Dermat. **3**:273, 1940.

8. Civatte, cited by Ott, H.: Psoriasis, Canad. M. A. J. **36**:165, 1937.

9. Lipschütz, B.: Untersuchungen über Dermotropismus: Theorie der Psoriasis vulgaris, Wien. klin. Wchnschr., 1910; Untersuchungen über Psoriasis vulgaris, Arch. Dermat. u. Syph. **127**:849, 1919.

10. Kyrle, J.: Vorlesungen über Histobiologie der menschlichen Haut und ihrer Erkrankungen, Wien, J. Springer, 1925-1927, Vols. 1 and 2.

11. (*a*) Gans, O.: Zur Histobiologie allergischer Hautbezirke, Arch. Dermat u. Syph. **151**:89, 1926. (*b*) Burks, J. W., and Montgomery, H.: Histopathologic Study of Psoriasis, Arch. Dermat. & Syph. **48**:479, 1943.

12. (a) Santori, G., and Ceccarini, F.: Ricerche sui rapporti tra psoriasi e ricambio lipidico: I. Stato attuale della questione ed osservazione statistiche, Gior. ital. dermat. e sif. **87**:429, 1946. (*b*) Simons, R. D. G. P.: Additional Studies on Psoriasis in the Tropics and in Starvation Camps, J. Invest. Dermat. **12**:285, 1949.

13. Charpy, J.: Recherches sur l'étiologie et de mecanisme du psoriasis, Bull. Soc. franç. dermat. et syph. **56**:460, 1949; Essai étio-pathogénique du psoriasis, Union méd. Canada **79**:1120, 1950. Barber, H. W.: Psoriasis, Brit. M. J. **1**:219, 1950. Selye, H., cited by Barber.

14. Reiss, F.: Psoriasis and Adrenocortical Function, Arch. Dermat. & Syph. **59**:78, 1949.

15. Riehl, G., Jr.: Zur Therapie der Psoriasis mit Nebennierenrindenextrakt, Arch. Dermat. u. Syph. **177**:252, 1938; Zentralbl. Haut- u. Geschlechtskr. **57**:491, 1938.

of the skin. I refer particularly to the careful examinations by Schamberg [16] and his co-workers, who believed that abnormal nitrogen metabolism and retention of nitrogen were present. Herrmann [17] found a retarded elimination of uric acid but no relation of this fact to clinical symptoms.

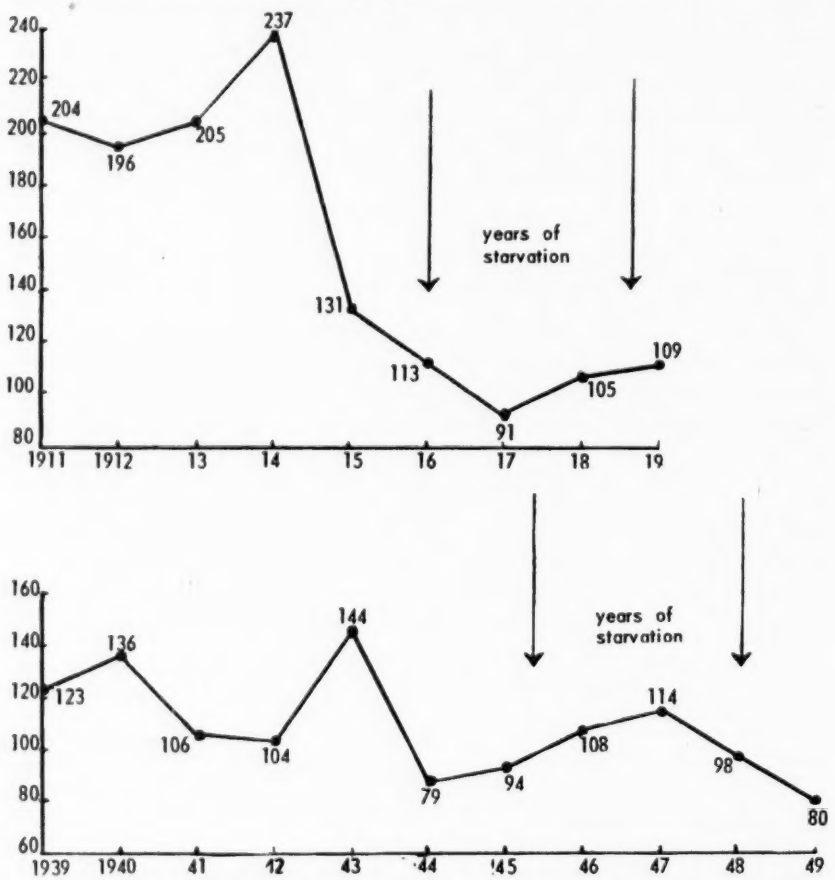

Fig. 1.—Graphs showing distribution of cases of psoriasis by years through the two world wars at the Clinic for Skin Diseases, University of Leipzig, from 1911 to 1919 (upper graph) and at the Clinic for Skin Diseases, University of Frankfurt am Main, from 1939 to 1949. There were 1,391 cases of psoriasis in men, women, and children seen between 1911 and 1919 at the University of Leipzig clinic. At the University of Frankfurt Clinic, 1,186 cases of psoriasis in men, women, and children were seen between 1939 and 1949.

Changes in the mineral metabolism (Hämmerli, Gaucher, Jamieson, and others) and the possibility of a connection between psoriasis and disturbances in the function of the endocrine glands have been discussed.

16. Schamberg, J. F., and Brown, H.: Studies of Inorganic Salt of the Blood in Psoriasis, Arch. Dermat. & Syph. **21**:737, 1930.

17. Herrmann, F.: Harnsäureuntersuchungen bei Psoriasis, Arch. Dermat. u. Syph. **161**:114, 1930.

Certainly, there may occasionally be disturbances in one or another endocrine gland, such as the hypophysis, adrenal glands, or thyroid, but this is not the solution to the problem of the cause of psoriasis. Glycosuria [18] and hyperglycemia [19] are mentioned, but Blast found the blood sugar content to be irregular, as did Leonhardi, Krause and I. Venturi [20] stated that there is a sugar storage in the skin of the psoriatic patient. Panti [21] found the relation of blood sugar to tissue sugar in the skin of the psoriatic to be increased. Königstein found no change in the function of the islands of Langerhans, i. e., the sugar metabolism is normal in the patient with psoriasis.

Fat and Cholesterol Metabolism.—The greatest effort was made in the examination of the fat and cholesterol metabolism. The most important impetus came from Grütz [22] and Bürger. In the main they found an increase in total fats in the serum of psoriatic patients; the total cholesterol was 50% higher and the phosphatide was also increased. Tolerance tests with administration of 5% cholesterol in 100 gm. of olive oil on fasting appeared to reveal, on the average, a high fasting value for total fat and cholesterol, followed by a slower rise in psoriatic patients than in normal subjects. In normal persons there were lower fasting values and a relatively quicker rise. The relation of free to esterified cholesterol, which is 1 : 2 in the normal person, approaches 1 : 1 and even 1 : 0.39 in the psoriatic patient. The mean phosphatide fraction (normally 0.3 mg. per 100 cc.) is usually much higher in the patients with psoriasis. Since in psoriasis these disturbances in the lipid metabolism are found to be so regular, Grütz suggested that this disease be classified with the lipidoses.

Grütz [22b] has tried to support this theory of psoriatic lipidosis by histologic examinations. He stated that a surprisingly large lipid content is often found in psoriatic lesions in the papillary body, as well as in the epidermis. According to the clinical type of eruption, there was lipid transport to the skin in varying amounts and types. He described the migration of large lipid-carrying cells, lipophages, from the papillary layer into the epidermis. Here these lipophages are found in clusters in the keratohyalin layer of the exudative and the pustular types of psoriasis. Grütz considered this another proof of the importance of the skin in lipid transport and elimination. At my hospital Steigleder examined the lipid content in psoriatic as well as in other diseased and normal skin stained with Sudan black B after the method of Lison and with Sudan III in 40% alcohol solution after the method of Grütz; altogether 34 cases were studied.

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18. Strauss, H.: Über neurogene und thyreogene Glykosurie, Deutsche med. Wchnschr., 1897, Nos. 18 and 20. Weinbrenner, F.: Ueber die Fälle von Psoriasis, welche in der königlichen Universitäts-Klinik in den Jahren 1879 bis 1894 beobachtet wurden, Inaug. Diss., Bonn, A. Henry, 1894. Gross, L.: Hautaffektionen bei Diabetes mellitus, Inaug. Diss., Erlangen E. T. Jacob, 1897. Nagelschmidt: Psoriasis und Pankreas, Berl. klin. Wchnschr. **45**:517, 1908.

19. Pick, W.: Blutzuckerbestimmung bei Psoriasis, Furunkulose und Lues, Dermat. Wchnschr. **72**:297, 1921. Raymond, cited by Nobl.[3b]

20. Venturi, T.: Contributo di osservazioni sulla patogenesi e su una nuova terapia della psoriasi, Dermosifilografo **13**:335, 1938.

21. Panti, A.: Osservazioni sul contenuto in glucosio della cute e su la terapia insulinica intradermica nella psoriasi, Dermosifilografo **15**:713, 1940.

22. (*a*) Grütz, O.: Die Stellung der Psoriasis im Rahmen der Lipoidosen, Verhandl. Gesellsch. Verdauungskr. (1938) **14**:81, 1939; (*b*) Neue histologische Beiträge zum Psoriasisproblem, Arch. Dermat. u. Syph. **177**:246, 1938.

Grütz distinguished four different types of lipid distribution: (1) that shown by the diffuse staining of the stratum papillare and stratum subpapillare; (2) that shown by diffuse staining plus migration of lipophages from the papillary and subpapillary layer into the epidermis; (3) that caused by the greater migration of lipophages; and (4) that in which the lipid content is the same as in normal skin.

Study of 10 control specimens, 5 of normal skin from healthy persons and 5 taken from persons with different skin diseases (basal-cell epithelioma, tuberculin reaction, acute dermatitis, a psoriasiform neurodermatitis, and eczematous mycosis fungoides), revealed that in any pathologic condition there is an increased migration of lipophages, and there is a difference only in degree from that seen in psoriatic skin. In seborrheic eczema the conditions are similar to those in psoriasis. In some cases there is no change, and in some cases there is great increase in migration of lipophages. Grütz's findings, therefore, are substantiated, but the histochemical changes are not typical for psoriasis. Levy-Franckel and Breant [23] and Versari [24] supported Grütz and Bürger. Ottenstein,[25] with Rost and later Incedayi and Moncorps, accepted the possibility of a connection between psoriasis and lipid metabolism in spite of the fact that they found only a slight increase in cholesterol on fasting but a more pronounced increase after the cholesterol-tolerance test. In their examinations hyperglycemia was found in 46% and the blood-sugar-tolerance curve was increased in 19%, which made Ottenstein suspect a dysfunction of the pancreas or the liver, i. e., of the carbohydrate metabolism. In treatment of 20 of 25 patients she was successful with a fat-free diet and small doses of insulin. Riehl,[15] Sartory, Hufschmitt, and Meyer,[26] and Gaté, Chanial, Vallet, and Humbert [27] reported more or less the same results, but the latter group emphasized that this disturbance in the cholesterol metabolism of the psoriatic patient is not the cause but only a side-effect of the disease.

Rothman [28] stated that the surface fat film of psoriatic patients has a lower ratio of esterified cholesterol to total cholesterol than does surface fat in normal persons. Possibly the psoriatic constitution is associated with weakened esterifying ability of

23. Levy-Franckel, A., and Breant, P.: De quelques anomalies plasmatiques et sérologiques chez les psoriasiques: Incidences thérapeutiques, Ann. dermat. et syph. **8**:109, 1948.

24. Versari, A.: Il ricambio dei grassi e la dieta povera di grassi nella psoriasi, Arch. ital. dermat. e sif. **12**:639, 1936.

25. Incedayi, C. K., and Ottenstein, B.: Report on Recent Researches Regarding the Relationship Between Psoriasis and Lipoidosis, Dermatologica **80**:18, 1939; Neuere Untersuchungen Über die Beziehungen zwischen Psoriasis und Lipoidosen, Acta dermat.-venereol. **21**:674, 1940. Zur Frage der Behandlung der Psoriasis mit kaliumarmer Diät und Nebennierenrindenextrakt, Dermatologica **80**:65, 1939; Diätbehandlung in der Dermatologie: V. Störungen des Kohlehydrat-Lipoid und Säuren-Basenhaushaltes (mit besonderer Berücksichtigung der experimentellen und statistischen Ergebnisse unserer Klinik); Neuere Untersuchungen über die Beziehungen zwischen Psoriasis und Lipoidosen, Deri Hastahklari ve Frengi Klingi, Ars. 5, No. 29, 1938.

26. Sartory, A.; Hufschmitt, G., and Meyer, J.: Recherches sur l'étiologie et la pronostic du psoriasis par l'étude du déséquilibre protido-lipidique du sérum, Bull. Acad. méd. Paris **119**:333, 1938.

27. Gaté, J.; Chanial, G.; Vallet, A., and Humbert, P.: Recherches sur la cholestérolémie dans le psoriasis, Ann. dermat. et syph. **9**:465, 1938.

28. Rothman, S.: Abnormalities in the Chemical Composition of the Skin Surface Film in Psoriasis, Arch. Dermat. & Syph. **62**:814, 1950.

the epidermis. Schaaf and Obtulowicz [29] did not find any worth-while difference between psoriatic and normal persons in the fat and cholesterol metabolism, nor did Rosen, Rosenfeld, and Krasnow [30] and Madden and others. My co-workers, Leonhardi and Krause, and I can only emphasize these facts (Fig. 2).

The following procedures were used in our studies:

1. Examination of total fat content was done by a modification of Bloor's method. The serum was extracted with a 1:3 mixture of ether in alcohol. It was then evaporated, absorbed in chloroform, filtered, evaporated, and weighed.

2. Estimation of free and, after a preliminary alkaline hydrolysis, of total cholesterol was done after the method of Schoenheimer and Sperry. The cholesterol was precipitated with digitonin, then estimated colorimetrically on the basis of the Liebermann-Burchard reaction.

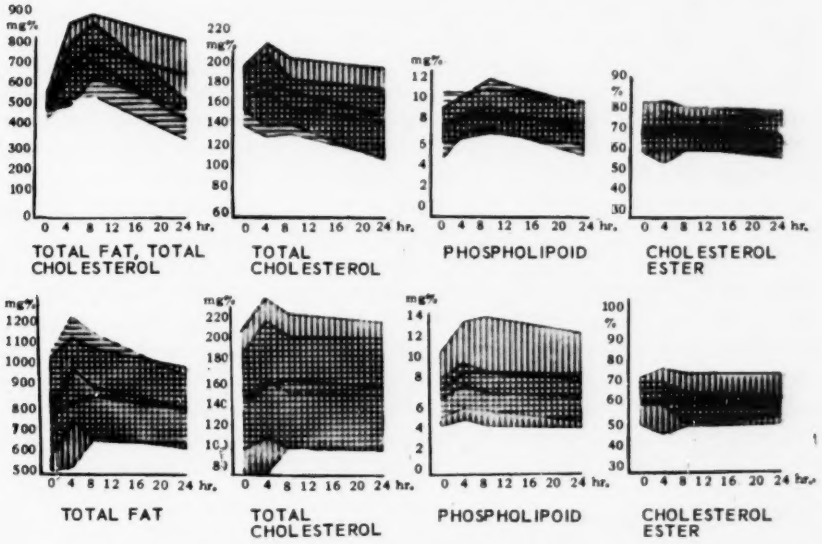

Fig. 2.—Charts showing average concentrations of lipid fractions in serum of psoriatic patients and controls on fasting and after fat intake (5 gm. of cholesterol in 100 gm. of olive oil). Upper group of charts is taken from work of Schaaf and Obtułowicz.[29] Lower group shows findings of Gans, Krause, and Leonhardi at the Skin Department, University of Frankfurt am Main. Portions with horizontal lines indicate individual variation in controls. Portions with vertical lines indicate individual variations in psoriatic patients. Dotted lines are probable average for controls; solid line, probable average for psoriatic patients.

3. Estimation of phospholipids was done after hydrolysis of the total fat extracts with sulfuric acid and 30% hydrogen peroxide. Colorimetric examination was according to the method of Fiske and Subbarow to determine the phosphorus released as phosphomolybdate, which is reduced to a blue dye with "eikonogen" (a preparation of sodium amino beta-naphthol-beta-monsulfonate).

29. Schaaf, F., and Obtułowicz, M.: Lipidstoffwechsel und Psoriasis, Arch. Dermat. u. Syph. **173**:200, 1935.

30. Rosen, I.; Rosenfeld, H., and Krasnow, F.: Studies in Psoriasis: I. Lipid Partition and Albumin-Globulin Ratio in One Hundred and Thirty Cases; II. Effect of Administration of Cholesterol (Tolerance Test) on the Lipid Partition and the Albumin-Globulin Ratio, Arch. Dermat. & Syph. **35**:1093, 1937.

In 10 psoriatic patients and 5 controls we examined total fats, total and free cholesterol, and phospholipids in the serum, on fasting as well as 4, 8, and 24 hours after oral administration of 5 gm. of cholesterol in 100 gm. of olive oil.

The lower part of Figure 2 shows the mean values on the individual analyses of the serum of the 10 psoriatic patients compared with those of the 5 controls. It shows that the fasting values of total fats were lower for the psoriatic patients than for the controls. After the intake of 5 gm. of cholesterol in olive oil, the rise in the level of total fats was slower in the patients having psoriasis compared with the controls. After 24 hours, the value for the psoriatic patients had not yet come down to the fasting level, whereas for the controls the value was well below the fasting level.

The examination of total cholesterol values showed analogous results. Contrary to this, the phospholipids in the fasting psoriatic patients were higher than in the controls. In the controls after the cholesterol-olive oil intake the maximum values were found after 4 hours, and after 24 hours the levels had come down to the fasting values. In the psoriatic patients, however, we found the maximum values 8 hours after the intake, and the levels had not yet returned to the fasting level even after 24 hours. The relation of free to total cholesterol (ester quotient) was lower in the psoriatic patients than in the controls and rose steadily for the 24 hours after intake, whereas the controls showed a diminishing tendency.

In spite of these differences, the mean values for the psoriatic patients were within the limits of the individual fluctuations in the controls. In agreement with the results obtained by Schaaf and Obtulowicz,[29] we found that in psoriatic patients the deviations in the lipid content in the serum after intake of cholesterol-olive oil show no great variation from normal. They are not significant enough to allow the conclusion that the psoriatic patient has anomalies in the lipid metabolism.

Simultaneously with these determinations, the sugar metabolism was tested by the Staub-Traugott method. We did not find any deviation from normal in psoriatic patients.

The vitamins, particularly vitamin C, were found to be decreased,[31] but no exact relation could be established between the intensity of the psoriatic rash and the vitamin C deficiency. Nevertheless, Lutz, Grüneberg,[32] and others reported good results with vitamin C therapy. At my hospital we did not see any worth-while effect either in psoriasis or in psoriatic arthritis, in spite of very high dosage and prolonged treatment. Some patients are cured; some improve in the long run, but many show no benefit at all. The situation appears to be very much the same as with fat metabolism and fat-free diet.

The reports of Bürger and Grütz considering psoriasis to be based on a disturbance in the cholesterol metabolism seemed to suggest the possibility of lipotropic compounds having an effect on the course of this disease. These compounds prevent a fatty degeneration of the liver in animals fed a diet rich in fats and poor in proteins. This can be explained by the fact that the lipotropic substances cause the transport of fat from the liver cells by development of lecithin. It was not possible to prevent

31. Reiss, F.: Psoriasis Vulgaris and Vitamin-C Deficiency, Indian J. Ven. Dis. **3**:240, 1937.

32. Grüneberg, T.: Hormonbehandlung in der Dermatologie unter besonderer Berücksichtigung des Psoriasisproblems, Med. Welt **11**:144, 1937.

development of cirrhosis of the liver (due to fatty degeneration or to arteriosclerosis) by feeding lipotropic compounds. Furthermore, it is well known that choline is primarily deposited in the skin.

We treated 12 psoriatic patients with average total doses of 135 to 315 gm. of choline or 74 to 116 gm. of methionine. In some cases this resulted in a distinct regression of the eruptions without any local treatment. This regression was particularly evident in the center of the lesions, even to the extent of the normal skin becoming visible, scaling and flattening resulting in the outer parts of the lesions. In no case, however, was there total disappearance of the lesions.

ETIOLOGICAL FACTORS

Disturbances of the endocrine glands have been mentioned by several authors. I have mentioned studies on the adrenal glands, hypophysis, and thyroid. Some facts hint at involvement of the endocrine glands, but there is certainly no connection

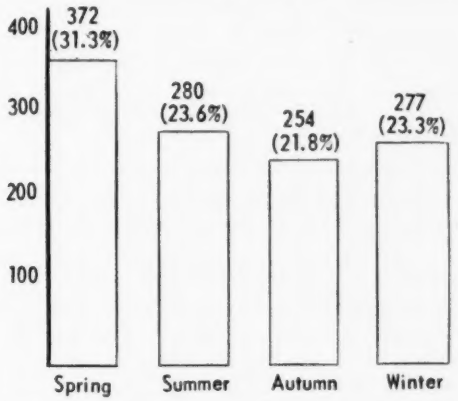

Fig. 3.—Graph showing seasonal distribution of cases of psoriasis over a period of 10 years. From the skin department of the University of Frankfurt am Main.

with the age of the patient. Psoriasis has been seen in a child 5 weeks old.[33] It is found in patients of all ages up to very old age, a fact which Kappesser[34] has reestablished from the statistics of my clinic.

Bettmann,[35] Moro, and others found an increase in psoriasis in spring and autumn, which made them suggest a connection with endocrine changes in these seasons. Such indirect proof is open to doubt, as all these statistics are based on figures difficult to assess. The fact, for example, that there was a regular increase of psoriatic inpatients at my hospital in winter and spring may possibly have to do more with the patient's being out of work and having no proper home, no heating, and inadequate clothing (Fig. 3).

A more definite relationship seems to be established in regard to the menstrual cycle in women. The first outbreak of psoriasis may coincide with the beginning of

33. Halle, H.: Säugling mit Psoriasis, Zentralbl. Haut- u. Geschlechtskr. **31**:558, 1929.

34. Kappesser: To be published.

35. Bettmann, S.: Zur Ätiologie der Psoriasis, Deutsche med. Wchnschr. **48**:762, 1922.

menstruation. During pregnancy the disease has been seen to improve [36] (by van Daal [36b] during each of nine pregnancies). On one occasion psoriasis reappeared in the third month of pregnancy, and a week later the woman had an abortion. On the other hand, pregnancy has had a provoking influence.[37] Such observations have been made by all of us. The first psoriatic eruption may also occur after the menopause.

To conclude, a connection between the function of the gonads and the first psoriatic eruption is generally accepted for all age groups from puberty to menopause, but there is no rule.

The nervous system has only rarely been blamed. The rarity of psoriasis in colored races in the tropics has been stressed.[12b] In India I found more psoriasis among the Parsees than among the Hindus or Mohammedans. The Hindus never eat meat, but they eat much oil and ghee (melted buffalo butter). Frequently a fat-free diet helped a Hindu who had psoriasis.

I have still to come across a report of a case of psoriasis in a colored person while he is living in a moderate climate.

Considering all these facts, one is reminded of Bloch's statement [38]:

> A few easily assessable elements of the blood are examined because this examination is at that time opportune and appears easy. A single condition in the complicated machinery of metabolism is separated and considered in isolation.

The insufficiency of such procedures has been proved again in this report. I believe that our methods of examination up to now have been too crude and that our research must set in at a much earlier, more primeval function of the body.

PATHOGENESIS

These rather disappointing results are mitigated by another group of observations which have been confirmed again and again. They refer to clinical facts, as well as to histological results. I stated at the beginning of the paper that a postulate for the possibility of a psoriatic eruption appears to be a potentially psoriatic person. The question, therefore, arises, how does such a person acquire this quality? There is general agreement nowadays that the quality is, firstly, a constitutional and hereditary factor which needs a trigger mechanism to become manifest. The second question, therefore, is, what is this trigger mechanism?

The constitutional factor is considered first. Many years ago, while I was working with Gruhle on the physical type of psoriasis patients, we found that there is no preponderance of the pyknic type, but that leptosome persons are comparatively much more rarely affected by psoriasis (Fig. 4). Grütz found that nearly one-third of 529 psoriatic patients had a family history of psoriasis, diabetes, or gallstones. This goes to prove that we must assume certain constitutional pathologic conditions in the psoriatic persons.

The constitution of a man to a great part is hereditary, and heredity has been found to play a major role in the history of the psoriasis. All statistics, including

36. (*a*) Spitzer, R.: Sklerodermie und Psoriasis bei Gravidität und Menstruation, Schles. derm. Gesellsch. **14**:2, 1925; Zentralbl. Haut- u. Geschlechtskr. **17**:270, 1925. (*b*) van Daal, I.: Een merkwaardig geval van psoriasis, Nederl. tijdschr. geneesk. **91**:2068, 1947.

37. Bonafos, M.: Psoriasis et grossesse, Gynéc. et obst. **45**:465, 1946. Spitzer.[36a]

38. Bloch, B.: Das Pigment, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, J. Springer, 1927, Vol. I, p. 434.

those of McKenna and Leven, show that it is a dominant trait.[39] At the Frankfurt Skin Hospital, Fürst [40] found 36% psoriatic persons in 103 family trees of 185 psoriatic patients. Elizabeth Hunt found 30%. Touraine [41] mentioned a dominant irregular trait. Thirty-two families with a common pedigree showed 172 psoriatic and 190 normal members, and, of these, 27 were carriers. Touraine concluded that possibly psoriasis depends on two factors. Both are present in families with and without carriers, or one of the factors is missing in these carriers, and, therefore, psoriasis does not become manifest.

I obtained another hint concerning the pathogenesis of psoriasis from a clinical observation. In a patient suffering from vitiligo the psoriatic eruptions started only on the vitiliginous parts. It was several weeks before the psoriasis papules

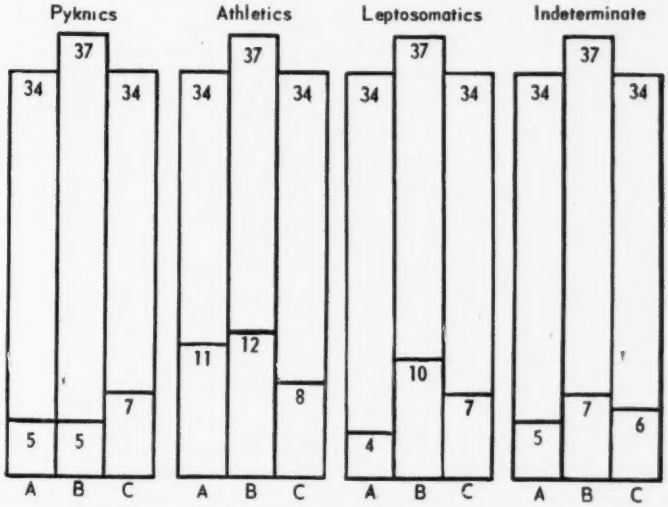

Fig. 4.—Occurrence of various "exact" constitutional types among persons with psoriasis (*A*), healthy skin (*B*), and various dermatoses (*C*).

began to appear on the normally pigmented skin. My search of the literature revealed a few observations like this by Kren,[42] by Riehl,[43] and recently by

39. Schamberg.[4] Bettmann.[35] Jordan, A.: Hundert und fünfzig Psoriasisfälle, Dermat. Wchnschr. **74**:445, 1922. Hecht: Vererbung bei Psoriasis und Keratodermie, XVI, Kongr. d. Deutsch. Derm. Gesellsch. Königsberg, 4. bis 10.8, 1929; Zentralbl. Haut- u. Geschlechtskr. **31**:411, 1929. Frenzl, F.: Psoriasis Vulgaris and Congenital Biologic Inferiority of the Skin, Česká dermat. **18**:161, 1938; **19**:15, 1939. von Heiner and Oppe. heim: cited by Parl, H.: Zusammenfassender Überblick über die heutigen Theorien von der Psoriasisätiologie, Zentralbl. Haut- u. Geschlechtskr. **28**:641, 1928. Hoffmann, E.: Über Psoriasis und das köbnersche Phänomen, Ztschr. Haut- u. Geschlechtskr. **9**:182, 1950.

40. Fürst, K.: Zur Vererbung der Psoriasis, Arch. Dermat. u. Syph. **153**:212, 1927.

41. Touraine, A.: Psoriasis familiaux, Bull. Soc. franç. dermat. et syph. **47**:219, 1940; Étude génétique du psoriasis, Ann. dermat. et syph. **2**:121, 1941.

42. Kren, O.: Vitiligo und Psoriasis, Wien. klin. Wchnschr. **38**:262, 1925.

43. Riehl, G.: Psoriasis und Vitiligo, Zentralbl. Haut- u. Geschlechtskr. **19**:716, 1926.

Troxell.[44] This coincidence seems to be very rare. From 1925 to 1950 I found only three cases. Obviously, something in the vitiliginous skin makes it different from normal skin and more easily accessible to the psoriatic agent.

What do we know about it to give us a clue? The decisive factor appears to be the lack of pigments, which, as we know from Bloch,[38] is due to the lack of an oxidase. Recently, Fitzpatrick, Becker, Lerner, and Montgomery [45] proved this to be tyrosinase and not dihydroxyphenylalanine (dopa). In vitiligo, too, there is a hereditary factor. Very often vitiligo is observed together with alopecia areata or alopecia totalis. In 10% of all cases of hyperthyroidism vitiligo and/or alopecia areata are found. To me this appears to be a fact of importance.

Stojanos and Martirenas and Robert [46] discussed the possibility of vitiligo being somehow dependent on the vascular system of the skin, not the nervous system. In half of their cases they found an increased basal metabolism, which also hints at a connection with the thyroid gland. They explained vitiligo as a consequence of a decreased oxidation process in certain vascular areas, whereby the less-resistant oxidase is destroyed. Haxthausen in his experiments on the pathogenesis of vitiligo attributes the disease [47] to influence of the nervous system; nevertheless, he states that it is not possible a priori to exclude local circulatory changes as the cause. The fact that in vitiligo the skin around the hair follicles, which is rich in blood vessels, is the last to lose its pigment points in the same direction. Hüllstrung and Ohlmeyer [48] found that in the vitiligo patient the pigmented, as well as the depigmented, skin has a tissue respiration only half that of a healthy skin, i. e., the oxygen consumption is only half of the normal.

In psoriatic and psoriasiform skin lesions the skin pigment—melanin—is found to be missing in varying degree in the epidermal cells of the basal-cell and the prickle-cell layers. There may be total lack of pigment or a decreased amount. According to our knowledge, this proves a disturbance in the oxygen metabolism as well as possibly the lack of the so-called prepigments and enzymes.

Returning to psoriasis and vitiligo, I should like to mention some work done at my hospital by Steigleder with the Nadi reaction of Gräff (with 20 cc. of phosphate buffer at pH 8.1) on psoriatic tissue that had not been fixed. The Nadi reagent contains α-naphthol solution and dimethyl-*p*-phenylenediamine.

The result of these studies was that in all cases of psoriasis (three of old lesions and nine of fresh ones) the acanthotic layer of the prickle-cell layer in psoriasis (and seborrheic eczema) appeared much more intensively stained with blue than the other parts of the epidermal epithelium. In other words, in the psoriatic lesion not only is there an increase in oxidase but this increase is restricted to the acanthotic parts.

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44. Troxell, E. C.: Psoriasis Developing in Areas of Vitiligo, Arch. Dermat. & Syph. **26**:1152, 1932.

45. Fitzpatrick, T. B.; Becker, S. W., Jr.; Lerner, A. B., and Montgomery, H.: Tyrosinase in Human Skin: Demonstration of Its Presence and of Its Role in Human Melanin Formation, Science **112**:223, 1950.

46. Robert, P.: Über die Vitiligo: Zugleich ein Beitrag zur Frage der Pigmentbildung, Dermatologica **84**:257, 1941.

47. Haxthausen, H.: Studies on the Pathogenesis of Morphea, Vitiligo and Acrodermatitis Atrophicans by Means of Transplantation Experiments, Acta dermat.-venereol. **27**:352, 1947.

48. Hüllstrung, H., and Ohlmeyer, P.: Über Atmung der überlebenden Haut bei Vitiligo, Ztschr. physiol. Chem. **280**:118, 1944.

According to Schümmelfeder,[49] the Nadi reaction depends on the presence in the tissue of an enzyme which is identical with the cytochrome oxidase of Warburg. Using the Barcroft method as modified by Warburg and Naegelein to establish the oxygen consumption in psoriatic tissue, I [50] was able in 1923 to arrive at the facts listed in the Table. These facts have been verified by Buhmann.[51] He pointed out, however, that the psoriatic skin contains a higher percentage of living epidermis than does the normal skin. After having corrected the values for normal and psoriatic skin, he still found an average increase in oxygen respiration of 27%.

Von Glasenapp has lately repeated these experiments at my clinic. She succeeded in separating the epidermal from the cutaneous part of the psoriatic skin. The results obtained tally fully with my former results and with those of Buhmann.[51]

Thus, there appears to be sufficient evidence that in psoriatic skin, particularly in young eruptions, there is an increased oxygen respiration. Oxygen consumption is also increased in some other skin diseases (neurodermatitis, lichenization) but not as much as in psoriasis.[51]

In vitiliginous tissue, we have found a reduced metabolism which manifests itself by a reduced oxygen consumption. Why is it that the psoriatic eruption

Oxygen Consumption of Diseased Tissue Compared with That of Normal Tissue

Type of Tissue	Quotient of Oxygen Consumption*
Recent psoriatic eruption	4.2
Old psoriatic eruption	2.6
Basal-cell carcinoma	1.94
Lupus vulgaris	1.54
Lupus verrucosus	1.1

* Quotient of oxygen consumption was obtained by dividing rate of oxygen consumption for diseased tissue by the rate for normal tissue.

develops here first? A possible explanation, according to Steigleder, is that in psoriasis, as proved by Nadi reaction and Barcroft method, cell respiration is increased. This may be considered a response of the body to the agent which causes the psoriatic eruption. If so, it is possible that psoriasis manifests itself first in tissue where cell respiration is lowered and because it is lowered.

In psoriasis, as well as in vitiligo, there is, as it were, no possibility left to develop pigment because the enzymes necessary for this are used elsewhere. This statement appears to be rather hypothetical, having as support only the increased oxygen metabolism and the Nadi reaction. It is put on a stronger foundation by the following facts which Steigleder has established in my hospital:

He has placed small pieces of normal skin in Ringer's solution (at body temperature) to which was added 1% triphenyltetrazolium chloride (TTC). As theoretically expected, an increased deposit of red formazan was found in just those spots in which normally the Nadi reaction is increased, i. e., near the hair follicle, sweat ducts, and sweat glands. I said this result was to be expected because

49. Schümmelfeder, N.: Histochemie der Zellatmung, Verhandl. Deutsch. path. Gesellsch., 1948, Vol. 32.

50. Gans, O.: Gewebsatmung und Röntgenwirkung, Deutsche med. Wchnschr. **49**:16, 1923.

51. Buhmann, A.: Atmung und Glykolyse in normaler und pathologisch veränderter Haut, insbesondere mit Hinblick auf Psoriasis, Biochem. Ztschr. **287**:145, 1936.

increased metabolism means not only increased oxidation but also increased reduction; in other words, a more active metabolism calls for more oxidases and for more reducing enzymes as well.

Figure 5 shows the formula for the process: Triphenyltetrazolium chloride by reduction is changed into red formazan:

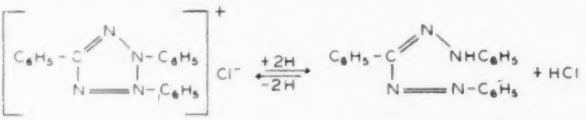

Fig. 5.—Structural formula showing process by which triphenyltetrazolium chloride is changed by reduction to red formazan.

Later, psoriatic skin lesions in discs 3 to 5 mm. thick were placed for half an hour in Ringer's solution at 37 C, pH 5.9.

Sixty specimens from 50 patients were examined; of these patients, 33 had psoriasis, 18 old and 15 recent psoriasis eruptions. From five psoriatic patients specimens of normal skin were obtained. Twelve patients had other skin diseases.

In general, the strongest deposits were noted in hair follicles, sweat glands and sweat ducts, basal layer, and surrounding epithelial layers.

In psoriasis, deposits were found within the acanthotic parts, that is, where Nadi-reaction spots are found. I should like to mention an important fact here. When several psoriatic lesions have been irradiated with ultraviolet light and the biopsy specimens taken after the erythema has developed, the formazan deposits are much less than in nonirradiated psoriatic papules of the same size from the same patient.

Of 29 patients examined, 22 showed a decrease and 7 an increase of the formazan deposit in the psoriatic lesion. This result, which may appear rather puzzling, can, however, very well be explained by referring to observations [52] I made some 25 years ago in regards to the pH of psoriatic eruptions. In psoriatic eruptions the pH was found to be more acid than in normal skin, a fact which can easily be proved with the use of neutral red dye. In fresh, not fixed, microsections of psoriatic skin the neutral red solution retains the red color longer than in the normal surrounding skin, where it turns yellow. Using the indicator method of Sörensen, I found a pH of 5.9 to 6.6 in the basal- and prickle-cell layers of psoriatic lesions. These differences remain as long as there is a psoriatic lesion visible on the skin.

We know from purely physicochemical processes that a change in the pH concentration may cause an acceleration, as well as a slackening, of chemical reactions, in particular those of oxidation and reduction. It appears, therefore, not too rash to conclude that for psoriasis the change to the more acid side is of some significance because it means a locally lowered metabolism.

Returning to the Nadi and triphenyltetrazolium chloride reactions, it is easy to see how well the results obtained fit in with this theory. In an acid pH, the deposit of formazan crystals is decreased; in alkaline pH, it is increased. Applying the results of these histochemical findings to the conditions of the living psoriatic skin, the conclusion may be allowed that here the oxidases and the reducing enzymes have to work under less favorable conditions than in normal skin owing to the

52. Gans, O.: Zur Pathogenese der Psoriasis vulgaris, Dermat. Wchnschr. **88**:280, 1929.

changed pH. Therefore more enzymes are necessary to guarantee the normal metabolism. Considering Steigleder's and my results, it may well be that the acanthosis and the sometimes tremendous increase in prickle cells are the morphological expressions of this effort. In tissues from other skin diseases which we have examined this is not so.

One last question is whether the shift in the pH of the tissue to the acid side and the reduction in oxidation are due to stasis in the capillary vessels, a fact which was first noted by Unna [1] and was confirmed by Burks and Montgomery and by me. This stasis in the papillary blood vessels of the skin in the psoriatic patient was verified in vivo by Bettmann [35] with capillary microscopy.

All these facts have been established on the basis of research methods which are generally recognized as reliable. Certainly, no one would like to consider as wrong all these results obtained by painstaking efforts. Why have we not yet gone further with our problem? What is the explanation for the many contradictory observations pointed out in the first parts of this paper? Either the results obtained are irrelevant to the problem or they solve or can solve only part of the problem, i. e., all the facts are only partially true in regard to the problem. Could it be that they are controlled by a common denominator which evidently has not yet been found?

LICHEN SIMPLEX CHRONICUS IN ORIENTALS

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IT HAS long been our impression that the incidence of lichen simplex chronicus is exceedingly high among the comparatively few Chinese patients seen in our practice, as contrasted with the occurrence among our white patients. There is scant mention in the literature regarding this observation. In 1936, Cleveland [1] analyzed 170 patients with lichen simplex chronicus in private practice. He states, "Although only 2.75 per cent of the total of 5,000 cases of diseases of the skin occurred in Orientals, 6.25 per cent of all the cases of lichen simplex occurred in Orientals." He felt that "the disease occurs more than twice as frequently in Orientals [Chinese and Japanese] as it does in members of the white race." Textbooks and articles on tropical diseases make little mention of lichenified dermatitis among Orientals. The literature is equally sparse in discussing lichenified dermatitis among Orientals in their native lands. Keim,[2] in reporting on the practice of dermatology in North China, notes "A strikingly large number of cases of chronic dermatitis are seen, mostly associated with lichenification." In addition, Fasal,[3] who worked in Malaya, states, "Circumscribed neurodermatitis was observed, mainly in Chinese."

Since our observations were limited to a relatively small series of Oriental patients, we wondered whether our colleagues who had more opportunity to observe these people had gained a similar impression. In order to obtain this information, the following letter was sent:

> It is our impression that the relative incidence of lichen simplex chronicus among the Chinese population is considerably higher than in the white.
>
> Since you have more opportunity to observe Orientals in your private and clinic practice, we are interested in gaining your impressions with regard to the following questions:
>
> A. Do you find a higher incidence of lichen simplex chronicus among the Chinese than among the white patients?

From the Department of Dermatology and Syphilology (Dr. Marion B. Sulzberger, Chairman), the Post-Graduate Medical School of New York University-Bellevue Medical Center, and the Skin and Cancer Unit of New York University Hospital.

1. Cleveland, D. E. H.: Lichen Simplex Chronicus: 170 Consecutive Cases Encountered in Private Practice, Arch. Dermat. & Syph. **33**:316 (Feb.) 1936.

2. Keim, H. L.: Impressions of Dermatology in North China, Arch. Dermat. & Syph. **17**:619 (May) 1928.

3. Fasal, P.: Cutaneous Disease in the Tropics: A Clinical Study Based on Observations in Malaya, Arch. Dermat. & Syph. **51**:163 (March) 1945.

B. Do you find that the neck is the area affected almost exclusively?

C. Can you suggest any causative factors for the increased incidence and localization?

The questionnaire was sent to about 110 dermatologists, and 100 replied. The statements made in the report are based on the experiences recounted in these letters.

RESULTS OF THE SURVEY

The majority of those who answered the questionnaire felt that they were in a position to offer only an impression of the incidence of lichen simplex chronicus among Orientals, since their small series of patients was insignificant statistically. There is, however, some significance to these observations when the results of all the individual questionnaires are analyzed. In direct response to the question concerning the incidence among the Chinese, 92% stated that there was a higher incidence, while 8% felt that there was no difference as compared with the rate of occurrence among their white patients. Fifty per cent of those who answered the questionnaire volunteered the information that the increased incidence of lichen simplex chronicus was not limited to the Chinese but was also observed among the Japanese and Filipinos. In fact, some stated that the Koreans, Hawaiians, and Mexicans were especially susceptible to lichenified dermatitis.

Dr. Harry L. Arnold Jr.,[4] of Honolulu, submitted the following detailed information on the occurrence of lichen simplex chronicus in his practice: During an arbitrary study period covering 15,000 patients, the diagnosis of lichen simplex chronicus was made by Arnold for 1,068 of them, an incidence of 7.1%. If one is to exclude the Caucasians, his average incidence among the dark-skinned races, composed mainly of Orientals, is 11.0%. This incidence is in contrast to the 3.4% rate of occurrence among the predominantly white patients studied by Cleveland [2] in this hemisphere and the 3.9% rate noted by Arnold among his Caucasian patients. The following is a tabulation of the findings of Arnold:

Race	No. of Cases of Lichen Simplex Chronicus	Per Cent of Cases	Per Cent of Office Population	Incidence According to Office Population
Japanese	379	35.5	19.9	12.7
Chinese	136	12.7	7.2	12.6
Koreans	31	2.9	3.9	5.3
Filipinos	165	15.4	3.9	27.5
Caucasians	327	30.6	56.1	3.9
Hawaiians	28	2.6	9.7	1.9

Arnold was able to give the incidence not only according to general island population but, more important, to "office population." Thus, in Hawaii, there is a threefold higher incidence of lichen simplex chronicus among the Japanese and Chinese as compared with the white race, and among the Filipinos there is an approximate sevenfold increase in incidence.

Harold M. Johnson [4] has had similar interesting findings in Honolulu. In reviewing his files for the past 10 years, he found that of all his patients with lichen simplex chronicus, 28% were Japanese, 27% were Filipinos, and 18% were Chinese. This is of special significance when one realizes that the 1950 population index is

4. Personal communication to the authors.

Japanese 34%, Filipinos 10%, and Chinese 5.9%. On this basis, the incidence among Orientals and Filipinos is considerably higher than among the Caucasians (exclusive of Spanish and Portuguese) in Hawaii.

Rees,[4] in reviewing the records of 750 completely unselected Chinese dermatological patients, found 60 (12.5%) with lichen simplex chronicus. Parker [4] found that 9 of 15 Chinese patients seen in recent years presented lesions of lichen simplex chronicus.

In contrast to the findings of Arnold, Johnson, Rees, and Parker and the impressions of many other observers in the United States is some statistical information from China. C. K. Hu,[4] in analyzing his data from the National Peiping University Medical College Hospital, noted an incidence of 3.3% of lichen simplex chronicus among 4,316 dermatological cases observed in a one-year period. This would suggest that the incidence is lower in the inhabitants of China than in the Chinese living outside their native land. Yang,[4] in reviewing approximately 30,000 cases of a variety of dermatoses at the National Medical College of Shanghai, found the incidence of lichen simplex chronicus to be 4%, this being quite similar to the findings of Hu from the National Peiping University Medical College Hospital.

In reference to the areas of localization, 50% of the observers noted the neck as being involved most frequently. Only five observers considered other areas to be the sites of predilection. The extremities, mainly the ankles, were next the most-frequently-mentioned areas. The genital region in the male was occasionally the only area of involvement. Rees [4] found the neck involved exclusively in 31 of his 60 cases, the neck plus other locations in 17, and other areas without involvement of the neck in 12. Hu [4] found the neck involved in 33% and the arms and legs each involved in 18% of the patients. Involvement of the extremities and the neck thus occurred in 70% of his patients. Yang [4] found the neck to be the site most commonly affected. Availability is, of course, one of the prime determining factors for the areas of localization, the nuchal area being generally accessible.

There was much speculation as to possible causative factors. The theories offered can be grouped mainly into the following categories: dietary (including allergic), traumatic, racial, and psychogenic.

Although some observers felt that the Orientals adhering to their original diet may lack vitamins, others pointed to a high incidence among Orientals who follow an occidental diet. Along this line, there is speculation as to the significance of a complete dietary change that these people may undergo when living in the Occident. Reiss [4] has done extensive intradermal testing with the protein fraction of silk and soybean sauce, with negative results. Novy [4] eliminated soybean and rice from the diets of his patients and felt that some of them improved slightly, but usually the majority did not respond to elimination of these two foods. Rice, which is one of the basic foods of the Orientals, is known to have a low allergic index. On the other hand, pork and fish are foods with a high allergic index, but elimination of these foods has usually been of no particular benefit. In contrast to the general impression that allergy plays a minor role, Wise and Ramirez [5] felt that a sufficient number of patients reacting to proteins were benefited by the removal of the

5. Wise, F., and Ramirez, M. A.: Protein Sensitization in Pruritus with Lichenification, Arch. Dermat. & Syph. **11**:751 (June) 1925.

offending protein (identified by skin scarification tests) to justify testing in all suspected cases. Yang [6] obtained positive family history of a similar skin eruption in some patients, but a family history of asthma, hay fever, or allergic rhinitis was decidedly rare. No conclusions can be reached as to the relationship between lichen simplex chronicus and atopic dermatitis among the Orientals. Contradictory impressions apparently have been gained by various observers.

Trauma was discussed by many observers as an important though not a determining factor. Items of clothing, such as high collars, jewelry, and especially wool garments, are considered mainly as trigger factors. Although contact irritants may play a role in causation and localization, the availability of the nuchal area for manual trauma was thought to be a more important factor.

Seven independent observers suggested that a racial predisposition exists among Orientals, with a tendency for lichenification of the skin to develop more easily as a reaction to various physical stimuli. Keim stated that in North China "a strikingly large number of cases of chronic dermatitis are seen, mostly associated with lichenification." Hu was of the opinion that in lichen simplex chronicus "there is no strong tendency indicating a hereditary factor." Brandt,[6] in noting an extensive list of constitutional characteristics which occur in cases of neurodermatitis, mentioned the prevalence of the disease in persons with dark hair and dark eyes. Cleveland noted that the disease occurs almost three times as often in brunettes as in blondes. Although the disease may have a predilection for dark skin, it is noteworthy that there is no increased incidence among Negroes, as compared with the white race. This was emphasized by Goldblatt in a discussion of Cleveland's paper in which he states, "I wish to call attention to the fact that in over twenty thousand Negroes examined, I have seen a case in only one, a young girl."

By far the majority of the observers, with a few exceptions, felt that the psychogenic factor is probably the most important in causing the development of lichen simplex chronicus in Orientals. It is a widely accepted opinion that psychogenic factors are important in this condition. English [7] believes that feelings of anxiety, hostility, inferiority, and envy produce emotional states that contribute neurophysiologically to skin changes. To what extent do these emotions exist in Orientals, and is the repression of emotions a contributing factor? We have repeatedly questioned Orientals who admit emotional conflict, in spite of a seemingly phlegmatic appearance. They begrudge the traditional training in stoicism received in their childhood and ask to be considered an emotionally sensitive group.

During World War II, the Japanese Americans, unfortunately, were placed in a difficult position, Kusayanagi [4] noted that the incidence of lichen simplex chronicus not only was high but increased markedly during the war years. She was able to observe the Japanese in war relocation camps, where, she states, the nervous tension was heightened greatly. During the evacuation, the Japanese were in doubt as to the future of their homes, family, and finances. Arnold [8] also noted a striking

6. Brandt, R.: Die Abgrenzung der Neurodermitis vom chronischen Ekzem auf Grund konstitutioneller Betrachtung, Dermat. Ztschr. **65**:330 (Feb.) 1933.

7. English, O. S.: Rule of Emotion in Disorders of the Skin, Arch. Dermat. & Syph. **60**:1063 (Dec.) 1949.

8. Arnold, H. L., Jr.: Incidence of Dermatoses in Office Practice in Hawaii, Arch. Dermat. & Syph. **53**:6 (Jan.) 1946.

difference between the prewar (2.0%) and the wartime (7.3%) incidence of lichen simplex chronicus in a practice which included over 40% Orientals.

Martin H. Orens [4] studied the results of this report from the psychogenic point of view and submitted the following information:

On the surface one must agree completely with your conclusions concerning the factors most important in the causation of the disease, but I must add a number of thoughts on the subject. I shall confine my discussion to the question of the Chinese, but most of what I will have to say would undoubtedly apply to the other Oriental races.

In this country there are at present a number of major subdivisions of the Chinese people. Firstly, there are those who were born in China and those who were born in this country. Secondly, we can divide both groups roughly into two major categories: those who are loosely called the intellectuals and who are here primarily for study purposes, and the workers, who go into a very few lines of work, mainly laundries, restaurants, and domestic work. It would be important to attempt to find out what the percentages of the disease are in all groups.

The most important work in the field of the Chinese in this country has been carried out by Research in Contemporary Cultures, which was started by Ruth Benedict in 1947. Much of my information was obtained personally from Dr. Ruth Bunzel of that group. Chinese people claim that they do not feel any sense of inferiority in this country but that they often feel discriminated against. This must be taken with a grain of salt, as it has been the general experience that most people who experience actual discrimination do show some feeling of inferiority. Chinese females in this country seem to show a greater ease of adjustment to the American way of life than do the males, and this is particularly in the field of showing of aggression, which is not allowed in the strict Chinese culture. Interestingly enough, in line with your finding about how often the neck is involved, the neck is always considered an important area for modesty, and it accounts for the high collars of Chinese women, who will not display their necks. This is also true, but to a less extent, of the legs.

It is a very common psychiatric experience that people with chronic skin conditions often appear on the surface to be extremely well balanced emotionally, and it takes a good deal of questioning to find out that the skin is the neurosis, and so no other symptoms are necessary; in other words, all conflicts which cannot be expressed otherwise are expressed in the body language of the skin. It is as if this organ becomes a safety valve of the emotional life.

It is extremely interesting to note that among Caucasians the incidence of the disease is higher among females, but this is not true among the Chinese. This fits in very well with the fact that the incidence of sexual difficulties is extremely high among the Chinese males both in China and in this country (probably more so here). It also fits in with the poorer adjustment that Chinese males make in this country compared with the females.

It would be impossible to make any very definite statements concerning the psychogenesis of the disease unless personality studies were made of a group of these patients with the disease, as compared with a control group without the illness. In general, it might be stated that the Chinese in this country are a minority race who experience definite discrimination but who are culturally trained not to show their emotions; the resultant conflicts, which are certainly greater here than in China, are exhibited in part as a cutaneous manifestation in the form of lichen simplex chronicus. It would be extremely interesting to have one of these patients undergo thorough psychoanalysis to see what could be found out as to why the skin is selected as the manifesting organ.

While the incidence of lichen simplex chronicus in the Caucasian shows a sexual difference, being more frequent in females, among the Orientals it is apparently as frequently observed in males. With regard to sex, Yang found the proportion to be 1:1, but slightly higher among the females. That impression was recorded by many observers. In the Filipino male a special problem arises, for few Filipino women migrate to this country. It was expressed well by Hall [4] "that the Filipino male, who tends to be diminutive genitally as well as congenitally, is likely to have

a difficult time with regard to sexual and family arrangement." Stokes,[9] in discussing the effect of nervous and emotional states on the skin, dwells at length on various sexual complexes that may contribute to the development of dermatitis.

SUMMARY

A significant number of dermatologists were queried independently, and the majority noted that the incidence of lichen simplex chronicus in Orientals in the United States is higher than among white patients. This confirms our original impression which prompted our survey concerning the occurrence of this disease among the Chinese. The higher incidence has been noted not only among Chinese, but among Japanese and Filipinos as well.

In addition to the many impressions, actual data are presented from Hawaii where large numbers of Orientals are observed. Arnold, in Hawaii, noted a threefold higher incidence of lichen simplex chronicus among the Japanese and Chinese as compared with the white race and among the Filipinos an approximate sevenfold increase in incidence.

The incidence of lichen simplex chronicus, as noted by Hu and Yang, in the native Chinese is essentially similar to the incidence among the white population in the United States, namely 3 to 4%. This is important factual evidence against the theory of any hereditary or constitutional tendency on the part of the Chinese toward development of this disease.

Possible reasons for the increased incidence of lichen simplex chronicus among Orientals residing outside of their native lands are discussed. There are adherents of dietary, allergic, traumatic, and racial theories, none of which has been proved definitely. Partially by exclusion but also by positive evidence, the psychogenic factors are recognized by most observers as being responsible for the production of this condition. Orientals themselves admit emotional stress, the outward signs of which they suppress by traditional training. Kusayanagi and Arnold both observed an even higher incidence of lichen simplex chronicus among Orientals during the difficult war years.

We feel that a study of this sort not only calls specific attention to the higher incidence of lichen simplex chronicus among Orientals outside of their native countries but strongly suggests that psychogenic factors are very important in causing the eruption.

9. Stokes, J. T.: The Effect on the Skin of Emotional and Nervous States, Arch. Dermat. & Syph. **22**:803 (Nov.) 1930.

Clinical Notes, New Instruments and Techniques

TREATMENT OF DARIER'S DISEASE (KERATOSIS FOLLICULARIS)

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Treatment in many cases of Darier's disease (keratosis follicularis) has been unsatisfactory. Although vitamin A therapy as reported by Peck and associates[1] is helpful, not all persons with the disease respond. It was in such a patient that the treatment of a daily bath with hypertonic saline solution, as advised by Gordon[2] for ichthyosis, was tried. It has relieved the itching, removed most of the hyperkeratotic tissue, and controlled the severe outbreaks.

REPORT OF A CASE

J. C., aged 28, first had the eruption eight years ago. There was no family history of skin eruption. Biopsy confirmed the diagnosis of keratosis follicularis. He had been receiving large dosages of vitamin A, 100,000 to 200,000 units a day, in oil and aqueous preparations with and without polysorbate 80 (monitan®). Locally, sulfur and salicylic acid ointments, vitamin A ointments, and x-ray therapy had been used. The last produced only temporary relief, and the others produced little change. For 30 minutes daily the patient was placed in a tubful of water containing 2 lb. (907 gm.) of rock salt (sodium chloride). These baths greatly improved his rash, reduced the amount of crusting, and alleviated the itching and discomfort.

This treatment does not produce a cure, for when the baths were stopped the lesions recurred. However, they have proved to be of great value in treatment of the condition.

1. Peck, S. M.; Glick, A. W., and Chargin, I.: Vitamin A Studies in Cases of Ichthyosis, Arch. Dermat. & Syph. **48**:32-34 (July) 1943.
2. Gordon, H. H.: Treatment of Ichthyosis, Arch. Dermat. & Syph. **52**:178-179 (Sept.) 1945.

URTICARIA PIGMENTOSA

Report of Case with Solitary Lesion

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Urticaria pigmentosa is a rare chronic skin disease characterized by pigmented macules, papules, or nodules which become edematous if rubbed. There are three varieties depending upon the predominant type of lesion: (1) macular, (2) papulonodular, and (3) maculopapulonodular. The macular variety is the commonest. Usually, the disease has its onset within the first year of life, and it frequently subsides at puberty. However, the disease may not occur until adult life. Initially, the lesions may be urticarial, or rarely bullous. The sites of predilection are the trunk, arms, and thighs, although any cutaneous area may be affected. A diagnostic sign is urtication of the lesions when they are rubbed or otherwise irritated by friction. A tendency toward generalized dermographism is frequently present. The degree of pruritus varies with the individual but is rarely severe. The only sequel is permanent pigmentation where the lesions have been.

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The individual lesions are discrete, circinate, or more usually elliptical, ranging from a few millimeters to several centimeters in the broadest diameter. In a given patient the size is fairly uniform. The color varies from yellowish-tan to a dull reddish-brown. They are usually numerous, although Little [1] reported a case with only 2 lesions; C. Fox [2] described another with approximately 12; and Gross [3] presented a patient with but a single lesion.

The histologic features are significant, consisting of an infiltrate of mast cells in the upper portion of the cutis and an increase of melanin in the basal cell layer of the epidermis. Mast cells are more numerous in children than in adults. It is generally accepted that they are derived from connective tissue cells that have acquired mast-cell granules. The mast cells are arranged in strands and columns in the papillae, adjacent to blood and lymph vessels, and around the sweat glands. There is also an infiltrate of polymorphonuclear leucocytes, lymphocytes, and plasma cells in the cutis. The epidermis and cutis are edematous, with capillary and lymphatic dilatation. Urticaria pigmentosa may be regarded as a potentially systemic disorder

Fig. 1.—Solitary yellowish-tan ovoid macule of urticaria pigmentosa on the extensor aspect of the right forearm of a 5-month-old infant.

and not solely a cutaneous disease, since mast cells have been demonstrated in the mesenteric lymph nodes, spleen, liver, and bone marrow.[4]

Treatment is unsatisfactory, although x-ray therapy has occasionally brought about temporary improvement.[5]

1. Little, E. G.: Urticaria Pigmentosa, Brit. J. Dermat. **17**:355, 393, 427, 1905; **18**:16-38, 1906.

2. Fox, C.: Urticaria Pigmentosa, in System of Medicine, edited by T. C. Allbutt and H. D. Rolleston, London, Macmillan Company, 1911, Vol. 9, p. 236.

3. Gross, P.: Urticaria Pigmentosa (Solitary Lesion), Arch. Dermat. & Syph. **29**:451, and 764, 1934.

4. Ellis, J.: Urticaria Pigmentosa, Arch. Path. **48**:426-435, 1949.

5. Fox, H.: Urticaria Pigmentosa, Arch. Dermat. & Syph. **7**:273, 1923. Michael, J.: Urticaria Pigmentosa, ibid. **9**:746-751, 1924. MacKee, G. M.: Urticaria Pigmentosa, ibid. **14**:98, 1926.

REPORT OF CASE

History.—L. N., a white girl aged 5 months, was initially examined in the dermatology clinic of New York Hospital in December, 1950. Her parents had noted a gradually enlarging, pigmented lesion on her right forearm since she was one month old. She was their only child. As far as they could ascertain the lesion was not pruritic, nor did it otherwise bother the infant. On two occasions it became bullous without any known trauma. Subsequently, the bullae had ruptured, liberating a clear, colorless fluid.

Physical Examination.—The abnormal physical findings were limited to the skin. On the extensor aspect in the proximal third of the right forearm was a solitary, yellowish-tan, oval, nontender, macule approximately 2.3 by 1.2 cm. in diameter. The lesion became urticarial upon rubbing.

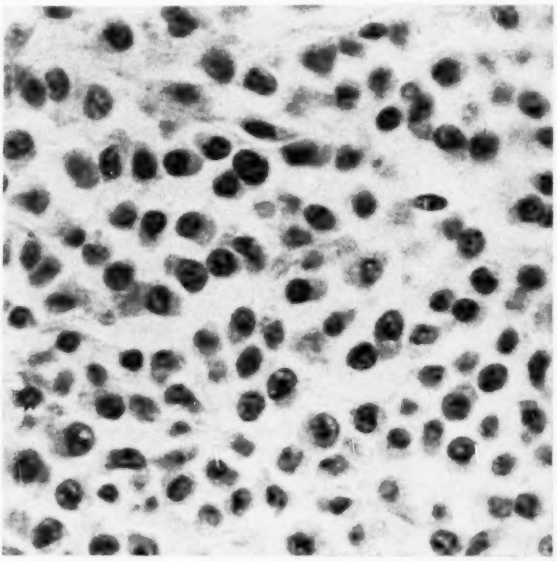

Fig. 2.—Histologic section demonstrating an infiltrate of mast cells in the upper portion of the cutis. The mast cells are large and cuboidal, with vacuolated cytoplasm.

Histologic Examination.—The epidermis showed alternate thinning and acanthosis with elongation of the rete pegs. The cutis was moderately edematous. An infiltrate of mast cells was present throughout the upper portion of the cutis, in the papillae, perivascularly, and around the sweat glands in the middle and deep cutis.

Clinical Course.—The patient was seen at frequent intervals for over one year. No new lesions developed during the period of observation, and the solitary lesion did not enlarge. On two additional occasions the lesion again became bullous without apparent cause.

SUMMARY

An instance of urticaria pigmentosa is reported in which only a solitary lesion was present. Both clinical and histologic findings were typical.

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MASKING OF FLUORESCENCE BY TETRACHLOROPARA-BENZOQUINONE ("SPERGON")

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In the treatment of tinea capitis, or ringworm of the scalp, caused by the microspora, the criteria generally accepted for clinical cure are loss of fluorescence, absence of organisms in smears or hairs, negative culture, and disappearance of clinical or gross evidence of a lesion for a period of at least two months following the discontinuance of all treatment. The patient is usually observed at weekly or biweekly intervals. As long as fluorescence is present, there is no absolute need for either a microscopic examination or a culture. With the disappearance of fluorescence, however, smears and cultures of the lesion become imperative.

Thick greases or ointments may easily mask fluorescence. In the routine examination of a patient's scalp, it should be impressed on the child's parents that the medicament must be washed out thoroughly before such an examination. Felsher,[1] for example, has found that 10 or 20% potassium iodide in fungicidal ointment, when massaged into the scalp, could disguise the fluorescence in one day. When the ointment was washed out, fluorescence returned in 12 to 24 hours.

Since the epidemic of tinea capitis became widespread throughout the United States, many new preparations to combat the disease have been put on the market. Unfortunately, none of these newer compounds has proved to be sufficiently effective per se not to require adjuvant forms of therapy. The most effective, when properly executed, has proved to be roentgen-ray depilation, particularly in those cases caused by Microsporum audouini. These newer preparations appear to work well in vitro but seem to lose their value when used in vivo. Apparently, the fungicidal agents are able to attack and in many instances kill off the fungi in the portion of the hair shaft which extends above the surface of the scalp, but they are unable to penetrate sufficiently into the follicle to affect the fungi present in that portion of the hair. In a number of busy private offices facilities for making and identifying fungus cultures are inadequate; consequently, the dermatologist relies on fluorescence, to a great extent, as an index of the progress of the infection. It is evident, therefore, that in most cases reliance on such a procedure may be very misleading.

One of the recent agents, a preparation containing 98% tetrachloro-para-benzoquinone as the active ingredient, is marketed under the name "spergon purified" by the United States Rubber Company. "Spergon" has been used effectively to control fungi attacking seeds. McGavack and his associates [2] have reported on series of patients treated with varying concentrations of tetrachloro-para-benzoquinone in various bases with apparently good results. Rankin, Dobes, Jones, and Alden [3] likewise claimed good results in patients having tinea capitis treated with this preparation. They found that when tetrachloro-para-benzoquinone was used in a 3% concentration, 28% of the patients were cured; with 20% concentration, 50% of the patients were cured, and with 40% concentration, 70% of the patients were cured.

Studies, observations, and reports from the Dermatological Department of the Barnard Free Skin and Cancer Hospital and the Department of Medicine, Division of Dermatology, School of Medicine, Washington University.

1. Felsher, Z.: Observations on the Fluorescent Material in Hairs Infected by Microsporon in Tinea Capitis, J. Invest. Dermat. **12**:139-144 (Feb.) 1949.

2. McGavack, T. H.; Wiener, D.; Cassio, A. L.; Bell, M., and Boyd, L. J.: Influence of Tetra-Chloro-Para-Benzoquinone in Human Fungous Infections, Arch. Dermat. & Syph. **59**:94-100 (Jan.) 1949.

3. Rankin, J. L.; Dobes, W. L.; Jones, J. W., and Alden, H. S.: Ringworm of the Scalp: Treatment with Tetrachloro-Para-Benzoquinone; Clinical and Laboratory Analysis, South. M. J. **44**:616-620 (July) 1951.

Potassium hydroxide mounts of Microsporum-audouini-infected hairs treated with tetrachloro-para-benzoquinone; × 145. *A*, nonexpanded hair showing spores and break in shaft. *B*, shows atrophic portion of hair above scalp surface, heavily encrusted with tetrachloro-para-benzoquinone, and follicular portion of hair shaft, heavily spore-laden.

PRESENT STUDIES

Because of the favorable comments made by several of the visiting dermatologists of the staff of the Barnard Free Skin and Cancer Hospital on the results obtained by treating tinea capitis with tetrachloro-para-benzoquinone, especially concerning disappearance of fluorescence, our resident dermatologists began the same form of treatment for a series of patients with tinea capitis. The complete results of the therapy will be published later by them. At present, one item of importance, the masking of fluorescence, is reported.

The patients were not selected but were treated as they came to the clinic. All had tinea capitis, and in each case Microsporum audouini was the causative agent. Each patient was treated with tetrachloro-para-benzoquinone, 30%, in the base polyethylene glycol 1500 (carbowax® 1500). The ointment was applied to the lesions twice daily. At the time of this writing, 17 cases have been seen, but for the purpose of this paper only 6 have been sufficiently worked up.

In these six patients fluorescence was noted to become less evident in a variable length of time. In three additional patients, fluorescence was much less noticeable in one, two, and six weeks, respectively. In the six patients, fluorescence disappeared in periods of two weeks, three weeks, two months, three months (two patients), and three and one-half months. In each of these six patients, the smears or microscopic examinations showed the organisms, and the repeat cultures also yielded Microsporum audouini.

Microscopically, the hairs above the surface of the scalp showed atrophic shafts covered with a yellow substance, the tetrachloro-para-benzoquinone (Fig. *A*). The hairs were irregular or rough in appearance and in many instances showed yellow crystals on the surface of the shaft. In addition, the hairs became brittle, showing several breaks along the shaft. In potassium hydroxide preparations (30%) the hairs did not expand as much as is customary. In those hairs on which the deposit of tetrachloro-para-benzoquinone was not too thick, spores could be seen with ease.

In spite of its incorporation in polyethylene glycol 1500, the tetrachloro-para-benzoquinone did not penetrate into the follicles, so that, although fluorescence of the exposed hair shafts was masked by the chemical deposits, the follicular portions showed no such deposits and consequently fluoresced brilliantly under the Wood light. The follicular portion of the hair shaft cleared well in the potassium hydroxide preparation, expanded, and revealed the usual fungus spores (Fig. *B*). In the discussion on the report made by Rankin, Dobes, Jones, and Alden [3] on the treatment of ringworm of the scalp with tetrachloro-para-benzoquinone, both Noojin and London observed that strong tetrachloro-para-benzoquinone ointments reduced fluorescence. London further remarked that hairs so treated and then observed in potassium hydroxide preparations showed spores in the roots, while the upper part of the hair was free of spores. Although this may be true in some instances, it does not apply, unfortunately, in all cases, since a heavy deposit of tetrachloro-para-benzoquinone will render invisible the spores that are present. On culture, however, viable colonies are obtained without difficulty.

In view of this experience of masked fluorescence, the dermatologist should be cautioned against relying solely on lack of fluorescence in lesions treated with tetrachloro-para-benzoquinone as an index of cure. Cultures and smears should be made, since, in the final analysis, the isolation of a living organism is prima-facie evidence of the existence of the disease.

SUMMARY

Infected hairs of tinea capitis caused by Microsporum audouini will show masking of fluorescence due to the deposits of tetrachloro-para-benzoquinone ("spergon") crystals. The hairs become brittle and show breaking points along the hair shaft. Potassium hydroxide preparations will not always reveal spores in the hair because of heavy deposits of the chemical. The drug apparently does not enter the follicle, since spores are readily visible on follicular portions of the hair. The dermatologist is cautioned against accepting lack of fluorescence in lesions treated with tetrachloro-para-benzoquinone as an indication of cure.

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KERION CAUSED BY TRICHOPHYTON RUBRUM

Report of Two Cases

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Although it is generally accepted that kerion due to Trichophyton rubrum is relatively rare, sporadic reports of this type of inflammatory reaction caused by this organism are appearing more frequently in the recent literature. The following is a report of two such cases.

REPORT OF CASES

CASE 1.—A 5-year-old white farm boy had been perfectly well until two weeks prior to his entry into the clinic, at which time he complained of pain in the back part of the neck while he was being bathed by his mother. She noticed a crusted, tender, swollen lesion in this area and instituted topical treatment at home with sulfur and lard. The lesion did not respond, and the child was taken to the family physician, who in turn applied penicillin ointment, with the same results. The lesion continued to enlarge; the child was brought to the Mayo Clinic. On examination he presented, over the occipital region, a palm-sized, slightly elevated, boggy, inflammatory mass, with many scattered purulent, draining sinuses. There were scattered erythematous, papular lesions over the trunk and arms and markedly enlarged but firm posterior cervical lymph nodes. There was partial alopecia in the area involved; many of the hairs were broken off near the scalp. Examination of the scalp with Wood's filter revealed no fluorescence. Specimens of the involved hairs were taken for direct examination and culture.

At mycological examination T. rubrum was isolated and identified on three separate occasions. On each observation scrapings from the scalp and hairs were directly examined in 10% solution of potassium hydroxide. Typical fungous elements were found throughout the skin cells, and the hair shafts were covered by spore mosaic. Cultivation on Sabouraud's agar and on an enriched medium gave powdery colonies with deep red reverse pigmentation which was visible around the edges of the top of the colony. A few macroconidia typical for T. rubrum were observed.

Treatment was instituted in an attempt to decrease the pain and inflammation; this was accomplished after two weeks of continued therapy. The patient also received 120 r of lightly filtered roentgen-ray therapy. After this, the lesion began to subside rapidly, although results of repeated culture were still positive for T. rubrum. At the last examination, one month after the institution of therapy, all inflammation had subsided, results of direct examination of hairs and the cultures were negative, and a new growth of normal-appearing hair was seen.

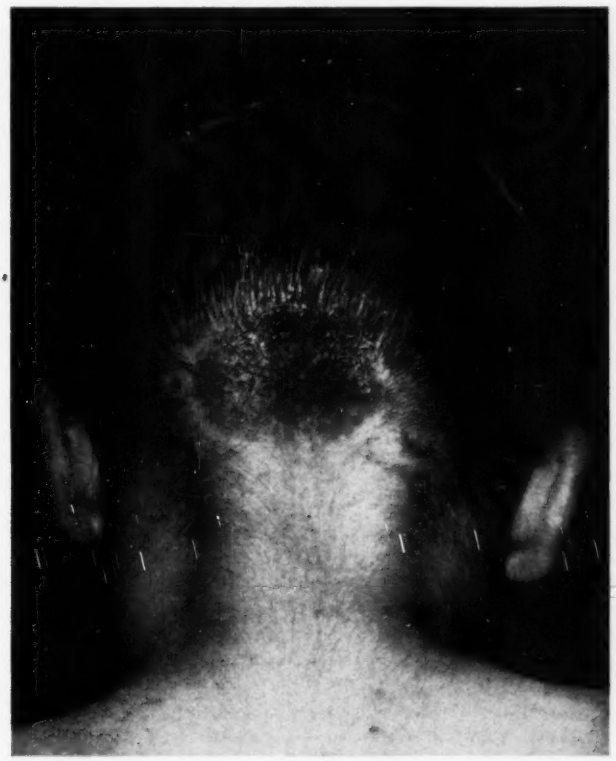
Comment on Case 1.—It was unfortunate that the causative organism was not suspected at the initial examination; if it had been, a photograph would have been taken. Only after much of the inflammation had subsided were we aware that we were dealing with an unusual situation. The photograph (Figure), therefore, does not reflect the true morphologic aspects of the kerion as we first observed it at the original examination.

CASE 2.—A 69-year-old farmer first came to the clinic on Aug. 30, 1951, because of a purulent granulomatous lesion 2 cm. in diameter at the base of the third finger on the dorsum of the right hand. The lesion, which had been present for three weeks, was said to have been precipitated by the penetration of a piece of straw. Regional lymphadenopathy was not present, but there were many small satellite pustules. Pus from these lesions and from the depth of the ulcer was taken for direct examination and culture. Examination of this material with Wood's filter did not demonstrate fluorescence.

Fellow in Dermatology, Mayo Foundation, University of Minnesota (Dr. Weber); Section of Biochemistry, Mayo Clinic (Dr. Ulrich).

The specimen for culture was received in the laboratory on a swab, so that direct microscopic observation of the material was not possible. The material, streaked on both Sabouraud's agar and an enriched medium, produced typical colonies of T. rubrum. Microscopic examination disclosed characteristics typical of those of T. rubrum, although only a few macroconidia were observed.

Topical therapy was administered; it consisted of wet dressings of a 1:8,000 solution of potassium permanganate and an antimycotic agent (propionate-caprylate mixture [sopronol® propionate-caprylate compound]). We were unable to obtain permission for photography. The patient left the clinic without further observation.



The lesion in Case 1, one week after the institution of treatment.

COMMENT

Loewenthal and Rein [1] recently reported a case in which tinea barbae of the kerion type had been produced by T. rubrum, and Wilson [2] reported a case in which trichophytic granuloma (tinea profunda) in the leg of a 54-year-old white woman had been caused by T. rubrum. As

1. Loewenthal, K., and Rein, R. L.: Tinea Barbae of the Kerion Type Produced by Trichophyton Purpureum: Report of a Case, A. M. A. Arch. Dermat. & Syph. **64**:194-196 (Aug.) 1951.

2. Wilson, J. W.: Trichophytic Granuloma (Tinea Profunda) Due to Trichophyton Rubrum, A. M. A. Arch. Dermat. & Syph. **65**:375-376 (March) 1952.

the commonest causes of kerion, Lewis and Hopper [3] listed infection with Microsporum lanosum, Microsporum audouini, Microsporum fulvum, Trichophyton crateriforme, Trichophyton gypseum, and Trichophyton niveum. We were unable to find any mention of kerion caused by T. rubrum.

The clinical characteristics of dermatomycosis due to T. rubrum usually are typical. They consist of a mildly inflammatory, dull red, superficial, scaly dermatosis of a chronic nature. The eruption is, for the greater part, rather resistant to the usual forms of topical therapy. The common sites of involvement are the feet, interdigital areas, the nails, medial aspects of the thighs, and the hands. Although the eruption tends to be localized, in some instances there may be generalized involvement. The condition in Case 1 in our report represents a localized inflammatory lesion with a generalized "id" reaction.

The favorable response and rapid healing in Case 1 further enhance the impression that the kerion type of inflammatory reaction tends to spontaneous cure.

SUMMARY

Two cases of the inflammatory kerion type of superficial mycoses are reported. In both instances the causative fungus was proved to be Trichophyton rubrum.

3. Lewis, G. M., and Hopper, M. E.: An Introduction to Medical Mycology, Ed. 3, Chicago, The Year Book Publishers, Inc., 1948.

CASE OF PITYRIASIS RUBRA PILARIS WITH LESIONS OF BUCCAL MUCOSA

JAMES MARSHALL, M.D.
JOHANNESBURG, SOUTH AFRICA

Lesions of the buccal mucous membrane are rarely found in cases of pityriasis rubra pilaris, and the classical authors, and many of those of the present day, either make no mention of such lesions or deny that they can occur.

A certain number of observations, some convincing and some less so, mention grayish or whitish taches on the buccal mucosa, resembling either syphilids or lichen planus.[1] In Jordan's case (cited by Sutton and Sutton [2]) there were two whitish, linseed-size, raised lesions on the palate.

The buccal lesions in the present case differed from those previously described.

REPORT OF CASE

V. H., a white South African aged 27, complained of an eruption of one month's duration. Nothing significant was noted in his personal or family history.

He had a typical eruption of pityriasis rubra pilaris, with plaques of little hyperkeratotic papules on an erythematous background on the dorsa of the fingers, elbows, forearms, nape of neck, ischial tuberosities, and thighs. There was a marked hyperkeratosis of the palms and soles.

The whole mucous membrane of both cheeks from the angles of the mouth to the last molars and into the depths of the pouches between gums and teeth was uniformly grayish-white and rough and had the texture and appearance of frosted glass. These lesions bore no resemblance to those of syphilis (at any stage) or of lichen planus (stretching the mucosa disclosed no streak or circle formation). The tongue, palate, and floor of the mouth were normal.

From the University of Pretoria.

1. Cohen, R.: Pityriasis rubra pilaire, in Darier, J., and others: Nouvelle pratique dermatologique, Paris, Masson & Cie, 1936, Vol. 7, p. 681.

2. Pityriasis Rubra Pilaris, in Sutton, R. L., and Sutton, R. L.: Diseases of the Skin, Ed. 10, London, Henry Kimpton (Hirschfield Bros., Ltd.), 1939, Vol. 1, p. 329.

Histological examination of skin from one forearm showed a moderate degree of hyperkeratosis, patchy parakeratosis, and plugging of the hair follicles. The granular layer was prominent, and there was acanthosis with hydropic degeneration of the basal layer. The corium showed a mild perivascular infiltrate consisting mainly of lymphocytes.

Treatment consisted of oral administration of vitamin A, 150,000 units daily, and the application of a keratolytic ointment twice daily. After three weeks there was a marked improvement. The hyperkeratosis of the palms and soles had almost vanished, and the papules had flattened in all the affected areas except on the dorsa of the fingers. The lesions on the buccal mucosa had entirely disappeared.

GRAYING OF HAIR FOLLOWING EPILATING DOSES OF X-RAYS

ISRAEL ZELIGMAN, M.D., D.Med.Sci.
BALTIMORE

During the past five years, I have had occasion to treat 58 private patients with tinea capitis. Of these, 56 have had Microsporum audouini infections and the other two have had the Microsporum lanosum type. Sixteen of these patients have been treated topically only, but only three have been properly followed until complete cure; one patient received a spot-epilating dose of x-rays with an excellent result. Of 41 patients who received a complete epilating dose, 37 were adequately followed until the complete return of noninfected hair. Several of the roentgen-epilated patients required additional manual epilation, but otherwise there have been no complications.

Of the 37 adequately followed roentgen-epilated patients, however, 3 have been brought back to my office because of the late appearance of gray hair where none had been noted by the parents or by me prior to the epilation.

REPORT OF CASES

Case 1.—D. P., a white boy aged 5 years, was first seen Oct. 18, 1948, because of scaly and bald spots on the scalp of four weeks' duration. Examination revealed a scaly plaque of incomplete alopecia 4 cm. in diameter over the vertex. With filtered long ultraviolet irradiation, numerous greenish fluorescent hairs were noted over the occiput, as well as in the plaque on the vertex. The culture was positive for Microsporum audouini, and a diagnosis of tinea capitis was made. On Oct. 26, a routine epilating dose of x-rays was given. This consisted of 365 r administered with the five-point equidistant technique, with the following factors: 100 kv., 10 ma., 10-in. (25.4-cm.) distance, and no filtration other than that inherent in the tube. The technique was successful, and defluvium ensued. Subsequently there was regrowth of noninfected light brown hair, and he was discharged as well March 24, 1949. On Sept. 14, 1949, the patient was returned for evaluation because of many gray hairs scattered diffusely over his scalp. There was a familial tendency to premature grayness, since both the patient's brother and his mother had become prematurely gray in their early twenties.

Case 2.—D. G., a white girl aged 6 years, was first seen Oct. 9, 1948, because of scaling and alopecia of the scalp of two months' duration. Examination disclosed five plaques of scaliness and incomplete alopecia over the vertex. With filtered long ultraviolet irradiation, many greenish fluorescent hairs were seen not only on the vertex but also on the occiput and in the area of the hair part. Culture was positive for Microsporum audouini, and a diagnosis of tinea capitis was made. The routine epilating dose of x-rays was administered Oct. 16, resulting in an excellent defluvium, followed by subsequent regrowth of noninfected light brown hair. She was discharged March 25, 1949. Several months later her mother noted numerous gray hairs diffusely scattered over the scalp, and this was corroborated by me July 6, 1950. The patient's mother had become prematurely gray at 17 years of age.

Case 3.—J. C., a white boy aged 6 years, was seen Jan. 25, 1949, because of scaliness and alopecia of the scalp of two weeks' duration. Examination in ordinary light and filtered long-

ultraviolet-wave irradiation disclosed a 2-cm. area of involvement on the occiput and a 3-cm. area on the vertex. Culture was positive for Microsporum audouini, and a diagnosis of tinea capitis was made. The routine epilating technique was administered Feb. 1, resulting in an excellent defluvium and subsequent regrowth of noninfected dark brown hair. He was discharged June 6, 1949. On May 24, 1950, he was brought back for examination because of the presence of diffusely scattered gray hairs. The patient's father had become prematurely gray at 18 years of age.

These three instances of grayness of hair following x-ray epilation were thoroughly inexplicable to me until I read an article by Chase [1] on the graying of hair experimentally produced in animals by roentgen radiation, and my study of the literature on this subject was thereby stimulated.

While performing other studies on colored mice with x-radiation, Hance and Murphy [2] observed the loss of hair pigment following epilating doses of x-rays to such mice. Subsequently, they were the first to study specifically the effect of roentgen irradiation on hair pigment of mice. These investigators, using 70 kv., 8 ma., 36-mm. target distance, and exposure times ranging from 20 to 100 minutes, exposed the shaved abdomens of black mice to x-radiation. Only white hair returned three weeks later to the abdomens of those mice exposed 50 to 100 minutes, whereas there was no alteration in the color of the hair on the irradiated abdomens of those animals that received less than 50 minutes' exposure. Similar but more detailed effects have been observed by others [3] on mice and rabbits.

According to Dry,[4] it is convenient to divide the developmental history of each generation of mouse hair into three phases. The anagen phase, the longest part of the growing period, lasts from the initiation of growth until, in colored hairs, pigment ceases to pass upward from the bulb at the base of the hair root. The second phase is the catagen phase, lasting only a few days, during which the root decreases in size. The final phase is the telogen phase, when there is no growth, the hair having become a clubhair.

Chase [5] found that inactive (telogen) follicles when x-rayed gave in the succeeding hair generation the maximum graying response for the particular dose. Active follicles (anagen) when x-rayed lost hair already present, and the succeeding generations were less gray for the particular dose than those from treated inactive follicles. He also found that the graying response was permanent; it persisted for succeeding hair generations. The effects of x-radiation on hair pigmentation of rabbits were also permanent, though the comparative sensitivity of telogens and anagens is still *sub judice*.[6] Mosaics (partially depigmented hairs) were also found in the various species studied.

COMMENT

Depigmentation of hair of patients following x-ray epilating doses was called to my attention by the parents of 3 of 37 adequately followed patients. All three had a family history of premature grayness in a parent (at 17 and 18 years of age and in the early twenties), and in addition

1. Chase, H. B.: Number of Entities Inactivated by X-Rays in Greying of Hair, Science **113**:714-716, 1951.

2. Hance, R. T., and Murphy, J. B.: Studies on X-Ray Effects: XV. The Prevention of Pigment Formation in the Hair Follicles of Colored Mice with High Voltage X-Rays, J. Exper. Med. **44**:339-342, 1926.

3. (*a*) Murray, J. M.: Studies on the Biological Effects of Roentgen Rays on Mice: I. Primary General Effects, Papers Michigan Acad. Sc., Arts & Letters **8**:401-409, 1927. (*b*) Danneel, R., and Lübnow, E.: Zur Physiologie der Kalteschwarzen beim Russenkaninchen: II. Der Einfluss von Röntgenstrahlen auf die Pigmentbildung, Biol. Zentralbl. **56**:572-584, 1936. (*c*) Lübnow, E.: Die Wirkung der Röntgenstrahlen auf die Pigmentbildung im Kaninchenhaar, Ztschr. indukt. Abstamm.- u. Vererbgsl. **77**:516-532, 1939. (*d*) Chase, H. B.: Greying Induced by X-Rays in the Mouse, Genetics **31**:213-214, 1946.

4. Dry, F. W.: The Coat of the Mouse, J. Genetics **16**:287-340, 1926.

5. Chase, H. B.: Greying of Hair: I. Effects Produced by Single Doses of X-Rays on Mice, J. Morphol. **84**:57-75, 1949.

6. Chase,[1] Danneel and Lübnow.[3b]

one patient had a brother whose hair had turned gray in his early twenties. My patients displayed gray hairs at 6 to 7 years of age; hence it is a reasonable presumption that they had the hereditary tendency to premature grayness and that this may have been hastened by x-radiation.

Rawles [7] has shown that hair pigmentation in the mouse is dependent initially upon melanoblasts migrating from the neural crest. Chase and Rauch [8] found that melanoblasts possess extensive dendritic processes which contain finely dispersed melanin granules and presumably "inoculate" matrix cells. Melanogenic dendritic cells were seen in the early anagen stage before the matrix cells acquired the granules. No melanoblasts, however, could be found in the x-ray-treated follicles producing white hairs. Radiation may thus destroy or inactivate the melanoblasts which supply the matrix cells of the follicle with pigment granules. They, therefore, reasoned that x-ray-induced graying, natural-age graying, and hereditary silvering in mice have features in common and may be the result of a failure in the melanoblastic source of pigment granules for each follicle.

The loss of hair pigment following x-ray epilating doses is, therefore, easily comprehensible and apparently may occur as a sequel to such treatment in persons with a hereditary predisposition to early grayness. The melanoblasts of such patients tend to lose their pigment granule formation function early, and x-radiation appears to hasten the decrease and disappearance of this function.

CONCLUSIONS

Three of 37 adequately followed patients who had received an x-ray epilating dose for tinea capitis displayed diffuse grayness of hair as a sequel.

All three of these children had a parent with premature graying of hair.

A review of the literature shows that depigmentation of hair of animals has been experimentally produced by epilating doses of x-radiation.

A hypothesis is submitted that hair melanoblasts with a hereditary predisposition to early loss of function are rendered ineffective even earlier by x-radiation.

1109 N. Calvert St. (2).

ADDENDUM

During February, 1952, the three patients above cited were seen again, and all showed an increasing grayness of the scalp hair.

7. Rawles, M. E.: Origin of Pigment Cells from the Neural Crest in the Mouse Embryo, Physiol. Zoöl. **20**:248-266, 1947.

8. Chase, H. B., and Rauch, H.: Greying of Hair: II. Response of Individual Hairs in Mice to Variation in X-Radiation, J. Morphol. **87**:381-391, 1950.

Correspondence

MAXIMAL TAR APPLICATION

To the Editor:—In a clinical note "An Effective Antipruritic Tar Application" (A. M. A. Arch. Dermat. & Syph. **65**:484, 1952), Dr. Thomas S. Saunders advises use of a solution of 10% coal tar solution with 20% tincture of benzoin in acetone for the treatment of anogenital pruritus. Perhaps it will interest Dr. Saunders and the other readers that a coal tar solution in acetone is widely prescribed in the Netherlands, Germany, Switzerland, and other continental countries (Siemens, H. W.: Allgemeine Diagnostik und Therapie der Hautkrankheiten, Berlin, Springer-Verlag, 1952). Well known is the so-called *Sacksche Pinselung*, a solution of 10% coal tar and 20% benzene in acetone. Solution coal tar, synonymous with liquor carbonis detergens, contains only 15% coal tar. (Why tincture of benzoin is added is not clear to me.) The pure coal tar in acetone is also generally well tolerated but is a bit messy. In Leiden, however, we use a benzene extract of coal tar ("liantral") in acetone, up to a concentration of 25%.

With this tar tincture we treat all types of pruritus anogenitalis, eczema, and psoriasis; we also use it on slightly inflamed and slightly excoriated areas, sometimes from head to foot. In 10 years we have not seen any intoxication or untoward reaction. Of course, there is always a small percentage of patients who acquire local skin irritations. Furthermore, as is well known, coal tar sensitizes against sunlight. For this reason, we treat the hands and face with pure solution coal tar (liquor carbonis detergens), which does not sensitize against sunlight.

Tar carcinoma, which theoretically can be expected, practically does not occur, as we know from the literature and our own experience, and thus is no contraindication to a maximal coal tar treatment. (We call this "maximal" treatment because pure coal tar is too messy to use on extensive surfaces; furthermore, pure coal tar is frequently irritating.)

Prof. H. W. Siemens,
Leiden, Netherlands.

RETICULOHISTIOCYTOMA OF THE SKIN

To the Editor:—Since the publication of our paper "Reticulohistiocytoma of the Skin" (A. M. A. Arch. Dermat. & Syph. **65**:701-713 [June] 1952), our attention has been called to the following articles which undoubtedly deal with the same dermatosis as was described by us.

Weber, F. P., and Freudenthal, W.: Nodular Non-Diabetic Cutaneous Xanthomatosis with Hypercholesterolaemia and Atypical Histological Features, Proc. Roy. Soc. Med. **30**:522, 1937.

Weber, F. P.: Cutaneous and Subcutaneous Nodules, with Juxtaarticular Infiltrations and Rheumatoid Symptoms, Brit. J. Dermat. **55**:1, 1943.

Weber, F. P.: A Syndrome of Rheumatoid Arthritis Combined with Multiple Xanthomatous Connective-Tissue Infiltrations, in Rare Diseases and Some Debatable Subjects, Ed. 2, London, Staples Press, Ltd., 1947, p. 39.

Portugal, H.; Fialho, F., and Miliano, A.: Histiocitomatosis gigantocitaria generalizada, Rev. argent. dermatosif. **28**:121, 1944.

Marcus R. Caro, M.D.
Francis E. Senear, M.D.
Chicago.

Society Transactions

DETROIT DERMATOLOGICAL SOCIETY

E. A. Hand, M.D., *President*

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R. H. Holmes, M.D., *Recorder*

Meeting held at Toledo, Ohio, Nov. 14, 1951

Hemangioma Treated with Thorium X. Presented by DR. HENRY A. BRUNSTING and DR. WILLIAM H. EYSTER JR., Toledo, Ohio.

J. H., a one-year-old white infant, with a birthmark involving the right side of the face, ear, and neck was treated with thorium X in the strength of 150 μc per cubic centimeter in alcoholic solution. Four applications were made (at one-month intervals), and marked blanching occurred.

DISCUSSION

DR. J. R. ROGIN: The method of choice is the use of CO_2 slush. This type of hemangioma disappears in time.

DR. F. R. MENAGH: Surgery is the method of choice.

DR. LOUIS BRUNSTING, Rochester, Minn.: I feel that most of these lesions will resolve spontaneously.

DR. HERMAN PINKUS, Monroe, Mich.: I do not believe that this type, the port-wine hemangioma, disappears spontaneously.

DR. ARTHUR E. SCHILLER: Whether there has been a change would be difficult to state from a comparison of the lesions, as they now are, with the color photographs. Color pictures are unreliable, for a reddish hue is given to all lesions. It does not appear that there has been much change.

DR. WILLIAM H. EYSTER, Toledo, Ohio: We have noted, during treatment, a progressive improvement in the lesions. These improvements have been manifested by a decrease in size, lightening of color, and disappearance of the discrete peripheral pseudopod-like portions of the hemangiomas.

Incontinentia Pigmenti. Presented by DR. HENRY A. BRUNSTING and DR. WILLIAM H. EYSTER, Toledo, Ohio.

J. S., a white girl 2 years of age, had a blotchy brownish pigmentation without previous abnormal reactions two weeks after birth. This spread from the left side of the trunk to the left arm and leg. There is no history of similar or related disorders in the immediate family. The mother is Rh-negative.

There is a bizarre distribution of chocolate-brown pigmented macules arranged in polyangular flecks, whirls, streaks, and patches limited to the left side of the patient. No atrophy or clinical evidence of other developmental abnormalities is present. On the right upper arm are a small area of dermatitis and adjacent areas of dermatitis with adjacent areas of depigmentation.

Histological examination by Dr. Carl Weller and Dr. Thomas Fitzpatrick, of the University of Michigan, was reported as follows: "There is an irregular and unequal distribution of melanin, with the chromatophores in this instance lying at the level of the basilar epithelium. A few histiocytes filled with melanin can be seen. This picture is compatible with the diagnosis of incontinentia pigmenti."

Temporary improvement is afforded by local applications of 33% monobenzyl ether of hydroquinone ("age-rite alba") in an ointment base.

DISCUSSION

DR. HERMAN PINKUS, Monroe, Mich.: I suggest the diagnosis of nevus unius lateris, because the distribution is linear and the appearance of whorls on some body areas is usual. There is no history of previous blistering or inflammation preceding the pigmentation.

DR. HENRY BRUNSTING, Toledo, Ohio: I agree that this case does not meet all of the criteria for this diagnosis. The arrangement and distribution are very suggestive. The lesions do not follow the lines of cleavage of the skin. There has been no unfavorable reaction to this topical therapy despite its continued use for eight months.

Localized Scleroderma. Presented by DR. HENRY A. BRUNSTING and DR. WILLIAM H. EYSTER JR., Toledo, Ohio.

H. C. R., a white man aged 64, has symmetrically distributed plaques beginning 13 months ago and now involving large areas of the neck, abdomen, back, and flexures.

The serum calcium is 9.5 mg. per 100 cc.

Cortisone therapy was started 11 months ago, and in seven days there was beginning resolution of the plaques. After three months of this therapy, 200 mg. daily of vitamin E (tocopherol) was substituted, and improvement has continued slowly to about 40% of the previous involvement.

DISCUSSION

DR. ARTHUR E. SCHILLER: I agree with the diagnosis. Why was a change made in therapy when there was improvement with cortisone?

DR. HENRY A. BRUNSTING, Toledo, Ohio: This was done for financial reasons.

Scrofuloderma Treated Topically with Aureomycin. Presented by DR. HOWARD J. PARKHURST, Toledo, Ohio.

B. L., an 8-year-old boy of Mexican ancestry, born in Texas, has deep, firm nodules on the neck, with irregular, deep scars, which appeared five years ago. Previous to treatment there was a profuse purulent discharge with crusting. The reaction to the tuberculin test was strongly positive. For five months he has had sun baths, a full diet with added polyvitamin therapy, and application of a 3% aureomycin ointment three times a day. Healing has progressed steadily and seems nearly complete.

DISCUSSION

DR. LOREN W. SHAFFER: Treatment with aureomycin ointment is a new method in my experience. It is surprising that calciferol (vitamin D_2) had not been used.

DR. SARTONO KERTOPATA, Djakartakotas, Djana, Indonesia (by invitation): We have been treating tuberculosis of the skin with streptomycin.

Discoid Lupus Erythematosus. Presented by DR. ARTHUR P. R. JAMES, Toledo, Ohio.

C. W., a white man aged 34, first had the appearance of typical lesions seven years ago. He was given bismuth, gold, and penicillin therapy, with little if any improvement. Three weeks of local application of cortisone ointment (25 mg. of cortisone in each gram of aquaphor® [an oxycholesterol-petrolatum ointment base]) appears to have reduced the activity, and subjective improvement is definite.

Discoid Lupus Erythematosus. Preesnted by DR. ARTHUR P. R. JAMES, Toledo, Ohio.

M. P., a white woman aged 20, has had typical lesions for 7 years. Bismuth therapy did not produce much improvement. Cortisone ointment (25 mg. of cortisone in 1 gm. of aquaphor®) produced some improvement in three weeks.

Discoid Lupus Erythematosus. Presented by Dr. Arthur P. R. James, Toledo, Ohio.

Z. R., a Negro woman aged 35, with typical lesions for six years, has received gold injections and bismuth by mouth and intramuscularly, with only slight improvement. Three weeks of local therapy with 25 mg. of cortisone to each gram of aquaphor® has produced a subjective improvement.

DISCUSSION ON DR. JAMES'S THREE CASES

Dr. Loren W. Shaffer: I accept the diagnosis as discoid lupus erythematosus in all three cases. While the patients have made rather remarkable improvement already, it is my feeling that the administration of cortisone should be made by internal means.

Dr. Louis Brunsting, Rochester, Minn.: The problem of treatment of discoid lupus erythematosus is a continuous issue, and there actually is no specific treatment. I have used 5% cortisone locally and did not note any improvement.

An oral report from Dr. A. P. R. James, Toledo, Ohio, four months later indicated that the improvement noted was transient.

Case for Diagnosis: Parapsoriasis en Plaques? Lymphoblastoma? Presented by Dr. Arthur P. R. James, Toledo, Ohio.

Dermatitis Herpetiformis. Presented by Dr. Henry A. Brunsting and Dr. William H. Eyster Jr., Toledo, Ohio.

Fox-Fordyce Disease. Presented by Dr. Henry A. Brunsting and Dr. William H. Eyster Jr., Toledo, Ohio.

Xanthoma. Presented by Dr. Henry A. Brunsting and Dr. William H. Eyster Jr., Toledo, Ohio.

Xanthoma. Presented by Dr. Henry A. Brunsting and Dr. William H. Eyster Jr., Toledo, Ohio.

Pityriasis Lichenoides et Varioliformis Acuta. Presented by Dr. Henry A. Brunsting and Dr. William H. Eyster Jr., Toledo, Ohio.

Localized Solid Edema. Presented by Dr. Henry A. Brunsting and Dr. William H. Eyster Jr., Toledo, Ohio.

Sarcoidosis? or Sarcoid-Like Tuberculoderm? Presented by Dr. Howard J. Parkhurst, Toledo, Ohio.

Pityriasis (Tinea) Versicolor of Tropical Type (Castellani). Presented by Dr. Howard J. Parkhurst, Toledo, Ohio, and Dr. Herman Pinkus, Monroe, Mich.

Dystrophy of the Hair, Nails, and Teeth. Presented by Dr. Howard J. Parkhurst, Toledo, Ohio.

Epidermolysis Bullosa Limited to the Feet in Father and Son. Presented by Dr. Howard J. Parkhurst, Toledo, Ohio.

E. A. Hand, M.D., *President*

C. A. Doty, M.D., *President-Elect*

C. J. Courville, M.D., *Secretary-Treasurer*

R. H. Holmes, M.D., *Recorder*

Jan. 9, 1952

Nevus Unius Lateris. Therapy? Presented by the Staff of Harper Hospital.

D. M., a Negro woman aged 22, since infancy has had verrucous hyperkeratotic papules and bands in linear arrangement, mainly localized to the left side of the chest and abdomen and the left lower extremities. The common laboratory examinations gave normal findings.

DISCUSSION

Dr. Frank Stiles Jr., Lansing: I suggest that the lesions be left alone.

Dr. James Rogin: I believe this is ichthyosis hystrix.

Dr. Arthur E. Schiller: I would use endothermy with the short wave current with the use of localized block anesthesia. We use a 1 mm. short-hop spark; but if the lesions are large we use the short loop. The lesions are cleaned off to the surface and lightly scarified with a sharp-pointed needle. The resultant scar can be further softened with carbon dioxide snow if necessary.

Dr. Arthur Curtis, Ann Arbor: I have seen several patients with excellent results after the use of sandpaper.

Lupus Vulgaris. Presented by DR. ARTHUR E. SCHILLER and DR. H. H. HOLMAN.

M. J., a Negro woman aged 67, noted a small "pimple" on her nose 14 years ago. This gradually spread to involve the nose, the upper lip, both cheeks, and the forehead. There is a granulomatous process, with considerable thickening and atrophic scarring of the forehead, cheeks, and upper lip. The distal half of the nose is bulbous and nodular with some erosion and fissuring. There are perforation of the bony nasal septum and bilateral corneal opacities.

The tuberculin test gave a positive reaction in a 1:1,000 dilution. A chest x-ray showed some pleural thickening in the costophrenic sinuses but no active pulmonary disease.

Gold therapy was of no benefit. Calciferol U. S. P. (drisdol®), 100,000 units daily, has produced noticeable improvement within five months, and vision has improved.

DISCUSSION

DR. H. H. HOLMAN: The soft nodules of the nose and the ophthalmological findings are suggestive of sarcoidosis. The scarring and granulomatous appearance of the upper lip speaks more for the diagnosis of lupus vulgaris. The picture is further confused by the septal perforation.

DR. THOMAS H. MILLER: I first saw this patient in 1931, when the lesions on the nose had been present "for some time." At that time the process consisted of multiple, discrete nodules, about 0.25 cm. in diameter, which eventually discharged a small amount of purulent exudate. Histological examination did not enable a positive differentiation between tuberculosis and syphilis. The patient was presented at the organization meeting of the American Academy of Dermatology and Syphilology, where the opinion was almost unanimously in favor of a diagnosis of syphilis. There was no response to treatment with oxophenarsine hydrochloride (mapharsen®) and bismuth subsalicylate, and very little improvement followed intravenous injections of gold sodium thiosulfate. Local destructive methods produced complete healing, but new lesions appeared within a short time. There was no involvement of the cheeks or forehead.

DR. ARTHUR E. SCHILLER: This patient was seen by us a number of years ago. The nose seems about the same size now, and the therapy has been unsuccessful.

DR. ARTHUR CURTIS, Ann Arbor: The findings are characteristic of lupus vulgaris.

DR. COLEMAN MOPPER: I favor the diagnosis of sarcoidosis because of the bilateral involvement of the eyes, with some corneal opacity. The bulbous appearance of the nose still present after all these years adds to the evidence.

DR. HERMANN PINKUS: The histological picture is compatible with either tuberculosis luposa or sarcoidosis. This difficulty in sharp differentiation is not infrequently encountered in Negro patients and is one of the strongest remaining arguments for a causal relationship between the two diseases.

I would not recommend surgical treatment at this time.

DR. C. A. DOTY: The possibility of leprosy should not be overlooked.

DR. JAMES ROGIN: The patient should be given a fair trial with calciferol.

Case for Diagnosis: Fixed Drug Eruption? Presented by DR. LOREN W. SHAFFER and the STAFF OF THE RECEIVING HOSPITAL.

L. H., a Negro woman aged 30, has had the development of an asymptomatic papular dermatitis in discrete pigmented patches over the bridge of the nose, on the right cheek, and at the right corner of the mouth within the past two months. The patches have become larger and darker. This patient takes feenamint® (laxative containing phenolphthalein) almost weekly. The last dose was taken 10 days ago. She was instructed to take some last night.

DISCUSSION

DR. KENNETH MOORE, Flint: A number of the lesions are typical of phenolphthalein eruption.

DR. CHARLES J. COURVILLE: The test dose last night produced no change noticeable at this examination.

DR. ARTHUR E. SCHILLER: The eruption first appeared on the legs and more recently in other areas.

DR. THOMAS H. MILLER: Are you certain the drug was taken last night?

DR. C. H. COURVILLE: She says she took it.

DR. H. H. HOLMAN: Antipyrine is as common an offender as phenolphthalein in the production of fixed eruptions. I suggest that the patient be given a provocative test with antipyrine.

Necrobiosis Lipoidica Without Diabetes. Presented by DR. C. J. COURVILLE.

Multiple Lentigines. Presented by DR. ALICE E. PALMER.

Sarcoidosis. Presented by DR. LOREN W. SHAFFER and the RECEIVING HOSPITAL STAFF.

Pruritus in a Diabetic. Presented by DR. C. K. HASLEY and DR. A. E. GULICK.

Recurrent Ulcers of the Legs. Presented by DR. ARTHUR E. SCHILLER and DR. H. H. HOLMAN.

Calcinosis Cutis Circumscripta. Presented by the STAFF OF HARPER HOSPITAL.

Syringocystoma. Presented by the STAFF OF HARPER HOSPITAL.

Neurotic Excoriations. Tendon Nodosities. Syphilis of Central Nervous System: Tabes Dorsalis. Presented by the STAFF OF HARPER HOSPITAL.

MANHATTAN DERMATOLOGIC SOCIETY

Samuel M. Peck, M.D., *President*

George M. Lewis, M.D., *Secretary*

Jan. 8, 1952

Keratosis Follicularis. Presented by DR. MAURICE J. COSTELLO.

F. A., a woman aged 51, has had an erythematous, crusted eruption of the face for 14 years. The lesions began as match-head-sized papules which crusted and by confluence presented the plaque-like lesions now seen. These originally covered the entire face, but there are now visible on the nose and nasolabial folds patches of normal skin. There are a few lentil-sized, brown, greasy, crusted papules on the outer third of the left clavicle. The diagnosis was confirmed by biopsy.

The patient has received vitamin A, 150,000 units daily, for the past 5 years. She states that the eruption always clears in the winter but recurs in the summer. The present eruption, which had its onset in November, is unusual for her.

DISCUSSION

DR. PERRY SACHS: I would suggest that a biopsy be performed on a palmar lesion to determine its exact nature.

DR. ORLANDO CANIZARES: The history of clearing up entirely is striking; I have never heard that before.

DR. PAUL GROSS: Dr. Eller presented a patient at the January 1952 meeting of the Section of Dermatology and Syphilology, New York Academy of Medicine, whose eruption also went into complete remission during the winter months. I think that keratosis follicularis is often aggravated on exposure to sunlight, and I interpret this as a Koebner phenomenon, similar to that seen in pityriasis rubra pilaris and pellagra.

DR. DAVID BLOOM: We have under observation at the clinic of the New York Skin and Cancer Hospital a woman with keratosis follicularis whose disease many years ago improved considerably after administration of vitamin A. When this patient comes to the clinic for some other cutaneous ailment, one can hardly see that she ever had keratosis follicularis.

DR. MAURICE J. COSTELLO: I suppose if I hadn't called attention to the patient's palmar lesion there wouldn't have been any discussion of this case. It is not known to many people that these lesions are characteristic of keratosis follicularis. That was called to my attention by Dr. Michelson, when he visited the New York Dermatological Society about a year ago. I have looked for them since and have practically never failed to find them. I agree with Dr. Peck that this disease thrives on the basis of photosensitivity, since the eruption is almost always on areas exposed to light.

Branchiogenic Cyst. Presented by DR. MAURICE J. COSTELLO.

W. H., a Negro woman aged 52, has had a gradually increasing, soft, cystic lesion in the right submandibular region for the past year and a half. She claims that this mass increases in size when she sings, or the intraoral pressure is increased. Aspiration of the contents of the swelling yielded yellow, gelatinous fluid, which contained no actinomycetes.

The hemogram showed 70% polymorphonuclear leucocytes and 30% lymphocytes. The jaw showed no roentgenologic abnormalities, but it was partially edentulous. The VDRL was 2 + on July 19 and 4 + on Nov. 8, and the Kolmer reactions 2 + and 4 + on the same dates. The Kline reaction was 3 + (titer, 1 to 2) on Nov. 6, and the Mazzini reaction, 4 +. The hemogram showed 14 gm. hemoglobin per 100 cc.; 5,800,000 erthrocytes, and 5,000 leucocytes, with 28% lymphocytes, 2% monocytes, and 70% polymorphonuclear leucocytes. The nonprotein nitrogen was 10 mg. per 100 cc., and the erythrocyte sedimentation rate was 16 mg. in one hour. The reaction to the Frei test was negative.

Treatment consisted of two fractional doses of x-rays, 100 r, administered on Aug. 6 and Aug. 13, without beneficial effect. Because of the doubtfully positive serologic reactions, 7,200,000 units of penicillin was administered, with no appreciable effect on the lesion. Surgical excision will be performed.

DISCUSSION

DR. WILBERT SACHS: The lesion appears to be situated rather high for a branchiogenic cyst; most are on the neck itself, and when the lesion extends upward there is usually a remnant on the neck. I do not doubt the diagnosis, but it is unusual.

DR. DAVID BLOOM: The appearance of the lesion so late in life is a factor against the diagnosis.

DR. MAURICE J. COSTELLO: Mixed tumors of the parotid are usually hard; this was soft and bladder-like. In the other cases of branchiogenic cyst of this type that I have seen the tumor was in this location. One patient was thought to have actinomycosis. The interesting feature is the apparent increase in size when the patient sings or otherwise increases the intraoral pressure.

Pyoderma Vegetans Successfully Treated with Chloramphenicol (Chloromycetin®). Presented by DR. DAVID BLOOM.

L. M., a Puerto Rican woman aged 50, was admitted to the dermatology department of Bellevue Hospital on Nov. 11, 1951, for an eruption of two years' duration.

A possible relevant factor in the past history was a bone abscess of the right ankle in early childhood. In 1935 she was operated on for a pilonidal cyst, which had been discharging pus since 1916. From 1926 on the patient had recurrent axillary abscesses. Five years ago she occasionally took bromo-seltzer® (acetanilid and sodium bromide) and other sedative drugs.

The eruption started four years ago with ulceration of the medial aspect of both arms, which left superficial scarring. Two years ago an ulceration developed in the groins and was treated by excision. At that time a small nodule also developed on the inner aspect of the left thigh, and this grew within two months to a raised, fungating mass, 5 by 2 in. (12.7 by 5 cm.) in diameter. Excision of this tumor, in 1950, was followed by partial healing, with a soft scar, but soon after a new, similar mass developed about the operative wound. The latter was excised in May, 1951. About three months ago similar lesions developed on the inner and anterior aspects of both thighs, on the buttocks, and on the right breast.

In the several months before her admission to Bellevue Hospital, the patient had been treated with penicillin and oxytetracycline (terramycin®) and with topical applications of aureomycin, bacitracin, and chloramphenicol ointment, without any beneficial effect.

The patient presented on the lateral aspect of the left hip, in the right groin, on the anterior aspect of the right thigh, and on the right breast well-defined, elevated, granulomatous masses of various sizes. The tumors were mostly oval, from 1 by 1.5 in. (2.5 by 3.8 cm.) to 5 by 2 in. (12.7 by 5 cm.) in size, reddish-brown or blackish, and surrounded by a purplish inflammatory halo. The surface was papillomatous and verrucous, covered with small abscesses discharging pus, and showing pitting and scarring in some areas. In the vicinity of the tumors and scattered over the trunk there were a few nodules and papulopustules, which apparently represented initial

stages of the tumors. On the left cheek was an indurated papulopustular lesion. Both axillas showed irregular scars. On the inner aspect of the left arm were oval and rounded soft scars, about 2 by 1 in. in size, with hyperpigmented borders and shagreen-appearing centers.

From her admission until recently, the patient ran a slight fever, with temperatures of 100.2 to 102.6 F. in the evening.

Roentgenograms of the chest were normal except for aneurysmal dilatation of the aorta. The urine gave a 1+ reaction for albumin on two occasions and on another contained 8.2 mg. of bromides per 100 cc. The blood cell and differential counts were normal, but the erythrocyte sedimentation rate was increased to 38 mm. in one hour. The hematocrit reading was 37%. Serologic reactions for syphilis were negative. The albumin-globulin ratio was inversed (2.8:5.8). Blood chemistry findings were otherwise normal. The cephalin flocculation test was weakly positive. The intradermal test gave a 1+ reaction to tuberculin in a 1:1,000,000 dilution, and a 2+ reaction to a dilution of 1:100,000.

Several biopsies at the New York Skin and Cancer Hospital, by Dr. C. H. Miller, and at Bellevue Hospital, by Dr. W. Director, revealed changes compatible with nonsyphilitic granuloma, or pyoderma vegetans.

Bacteriologic examination at the Skin and Cancer Hospital showed two organisms: (1) numerous hemolytic colonies of non-lactose-fermenting, Gram-negative rods, identified as Proteus mirabilis, and (2) colonies of nonhemolytic streptococci, which were isolated in subculture and identified as the enterococcus type of Streptococcus. The same organisms were found at Bellevue Hospital, in addition to Micrococcus pyogenes var. albus. Sensitivity tests of these organisms revealed that P. mirabilis was highly sensitive to chloramphenicol, moderately sensitive to streptomycin, and resistant to penicillin, aureomycin, and oxytetracycline. The enterococcus was highly sensitive to penicillin, aureomycin, streptomycin, and oxytetracycline, and moderately sensitive to chloramphenicol. The M. pyogenes var. albus was moderately sensitive to penicillin and resistant to aureomycin, streptomycin, and oxytetracycline.

Since Nov. 26 the patient has received 2 gm. of chloramphenicol daily. On Dec. 5 she was given in addition 300,000 units of penicillin daily, as well as topical applications of potassium permanganate solution and chloramphenicol ointment. Oxytetracycline was later substituted for penicillin. On Dec. 6, about 12 days after institution of treatment with chloramphenicol, the lesions began to show diminution in the discharge and some central flattening, and since then a gradual cessation of purulent discharge and flattening of the lesions have taken place. At present most lesions are almost entirely flat, and some show a raised papillomatous surface in places.

DISCUSSION

DR. GEORGE C. ANDREWS: This case has been thoroughly studied, and I congratulate the presenter. I wonder why this patient is sensitized to such ordinarily nonvirulent bacteria, for Proteus and M. pyogenes var. albus usually do not cause much trouble. It has been frequently stated that sensitivity tests are not reliable, but I use them all the time and think them very helpful.

DR. MAURICE J. COSTELLO: I saw this patient at the Bellevue Hospital dermatological staff conference, and there has been remarkable improvement since then. A diagnosis of tuberculosis was entertained because of the lesions in the axillas, the multiple involvement, and the course. The result in this condition is remarkable.

DR. ANTHONY C. CIPOLLARO: A few years ago we would say that pyoderma vegetans was caused by the microaerophilic bacillus of Meleney. The bulk of these infections are clearing up with specific antibiotics. We have failed to cure many of these infectious granulomas with antibiotics because the drugs have been selected at random. Specificity tests should be made with the causative organisms.

DR. PAUL GROSS: Patients with this condition usually have one of two predisposing diseases: Many have debilitating ulcerative colitis, but I have seen several cases in which the pyoderma was preceded by osteomyelitis. The patient presented here gives a history of a bone abscess many years ago. Chronic granulomatous and ulcerative lesions of the skin produced by pyogenic organisms represent an unusual occurrence; as a rule, pyogenic bacteria produce acute lesions of the skin.

DR. DAVID BLOOM: Examinations were made for aerobic and anaerobic bacteria, and no microaerophilic organisms were found. This patient probably does not owe her recovery to treatment directed against the enterococcus, because before she came to us she had received, without benefit, oxytetracycline and penicillin, to which the enterococcus is sensitive. Great improvement began soon after chloramphenicol was given, and the Proteus organism was found highly sensitive to that drug. From previous experience we learned that vaccine is of benefit, but in this case the bacteriologist could not produce a vaccine because elimination of virulence would require so much heating that it would kill the antigen also.

Chronic Radiodermatitis. Presented by DR. MAURICE J. COSTELLO.

P. M., a man aged 40, received nine x-ray treatments for sycosis vulgaris in 1937. This was followed by a nine month course of weekly x-ray therapy by a private physician. In 1939 the patient began to note the changes which are now present. He was treated with alvagel® but became sensitive to the medicine, so its use was discontinued. At present the patient is being referred to the department of plastic surgery for operation.

The patient presents depigmentation, pigmentation, atrophy, telangiectasia, and alopecia of the chin, cheeks, forehead, and eyebrows. The skin of the forehead shows moderate erythema.

DISCUSSION

DR. PERRY SACHS: This case would warrant an extensive operation. I wonder whether such a procedure could be performed in the presence of active follicular lesions.

DR. ORLANDO CANIZARES: I am wondering how the graft will take.

DR. ANTHONY C. CIPOLLARO: The graft should take; one of our colleagues had all the skin removed and has a functioning mouth and face. I think this patient still has a few years ahead of him; he has many keratotic and sclerotic areas and would probably be better off with some fresh skin in place of what he now has.

DR. MAURICE J. COSTELLO: I have known this man for a number of years, in the hospital and at the clinic, and I suggested that he have a plastic operation long ago. I recently had a patient in whom severe radiodermatitis developed, one of those Tricho Institute cases, and sent her to a general surgeon, who is a very clever man. He took several pieces of skin as a split-thickness graft from the abdomen and spent about four hours in the operating room, with an excellent cosmetic result. I don't think you have to carry out a complete procedure in this case; you could do the upper part first and the lower later.

Boeck's Sarcoid. Presented by DR. MAURICE J. COSTELLO.

S. R., a Negro aged 38, has been having numerous papules on the right ala nasi at the vermilion border for 1½ years. These papules increased in size and number until a granulomatous lesion developed. He also had some pinhead to match-head-sized papules on the vermilion border of the right side of the lower lip. A specimen from a granuloma of the nose was submitted to biopsy, and the remainder of the lesion was electrodesiccated. The biopsy showed typical Boeck's sarcoid.

The tuberculin test was negative. A Kveim test was performed on Nov. 27, 1951, and on last examination a split-pea-sized papule was palpable.

DISCUSSION

DR. ORLANDO CANIZARES: At the Veterans Administration Hospital a few patients with sarcoidosis have been treated with cortisone. It is thought there that about half the patients will improve with therapy.

DR. ANTHONY C. CIPOLLARO: I have never used cortisone for sarcoidosis, and I have no specific treatment to offer. In most cases of sarcoidosis recovery is spontaneous.

DR. PAUL GROSS: It should be stressed that this is a male patient and that sarcoidosis, at least among Negroes, is comparatively rare in males.

DR. DAVID BLOOM: The eruption in the Negro which is called sarcoidosis, but which I consider to be tuberculosis, is encountered as often in men as in women.

DR. GEORGE C. ANDREWS: I should not be inclined to give cortisone; it would probably clear up the lesions, but when its use was stopped the disease would come back. I should prescribe cortisone ointment. The eruption being in his nostrils, some of the cortisone would probably be absorbed.

DR. WILBERT SACHS: If one assumes that sarcoidosis is due to tuberculosis, what is the chance of lighting up the original tuberculous focus by giving cortisone?

Samuel M. Peck, M.D., *President*

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Feb. 12, 1952

Cheilitis Factitia. Presented by DR. DAVID BLOOM.

J. C., a boy aged 8 years, was seen at the New York Skin and Cancer Hospital for the first time on Jan. 18, 1952, with an eruption on the lower lip, consisting of several lesions inside the lip, near the angles of the mouth, which appeared as grayish, papillomatous tumors. The mother stated that these lesions had been appearing on and off since May, 1951. About two weeks later the patient was seen with a new lesion located on the inner aspect of the right side of the lower lip, consisting of a necrotic white streak. On questioning, the child admitted being in the habit of picking and pulling off the mucous membranes of the lower lip. Biopsy of a lesion was reported as showing organizing granulomatous tissue.

DISCUSSION

DR. WILBERT SACHS: It may be cheilitis factitia, but I can't rule out a diagnosis of Sutton's disease. If I did not know the history, that is the diagnosis I should make.

DR. PERRY SACHS: The boy claims he just picks his lips. However, microscopically the granulation tissue extends into the deep cutis, a feature which favors the diagnosis of leukoderma acquisitum centrifugum (Sutton's disease).

DR. GEORGE M. LEWIS: It is sometimes difficult to get a straight history from a child of this age, as the tendency is to agree to leading questions. The condition does not impress me as essentially factitial, although there are traumatic features. Neurodermatitis must be considered, as many cases of cheilitis have been proved to my satisfaction to be this.

DR. DAVID BLOOM: When we first saw him, the boy presented peculiar raised, papilloma-like lesions, which could not be diagnosed. Several days later he had a similar lesion of recent origin, linear in configuration, which strongly suggested a factitious element. He then admitted to picking and pulling the mucous membrane of the lower lip.

NOTE: After further observation, we came to the conclusion that the factitious element was superimposed on aphthous lesions.

Case for Diagnosis: Leukemid? Dermatitis Medicamentosa? Presented by DR. ORLANDO CANIZARES.

E. W., a man aged 34, is presented from the Veterans Administration Hospital, Bronx, N. Y., with an eruption on the trunk and extremities of one month's duration.

An operation for a herniated nucleus pulposus was done on Dec. 19, 1951, and procaine penicillin was given postoperatively until Dec. 23. The patient was also given acetylsalicylic acid, codeine, and secobarbital (seconal®) sodium. An erythematous eruption began to develop on Jan. 10, and by Jan. 13 there was a clear-cut urticaria. All medication was discontinued at this time, and the patient was given diphenhydramine (benadryl®) hydrochloride. By Jan. 15 he was febrile and complained of joint pains, and the urticarial lesions on the lower extremities showed a hemorrhagic tendency. He was inadvertently given acetylsalicylic acid and phenobarbital on that day. He continued febrile until Jan. 17, and the joint pains subsided the next day. The urticaria has continued, with several exacerbations, and he still has some scattered urticarial lesions. Erythematous macules and papules have also appeared on the chest in the last two weeks. He feels well and has no complaints other than back and leg pains.

There are large oval, pigmented, flat plaques on the legs, residuals of the previous hemorrhagic urticarial lesions. A few erythematous urticarial wheals are seen on the arms and thighs, and discrete erythematous papules are scattered across the chest.

Blood counts in this case are listed below.

Date	WBC	Pmn., %	Lymph., %	Mono., %	Eos., %	RBC, Millions	Hgb., Gm./100 Cc.	Thrombocytes
11/14	4,600	62	36	2	..	5.06	14.8	.....
12/18	3,250	49	39	11	1	4.65	14.8	.....
1/13	3,000	..	..	..	..	4.95	....	.....
1/15	2,600	35	59	4	2	3.90	13.0	115,000
1/17	1,450	34	58	3	3	4.15	14.2	.....
1/18	2,250*	29	57	2	4	3.83	12.8	.....
1/22	1,500†	27	59	2	6	4.18	12.4	54,340
1/31	1,800‡	20	64	1	5	3.86	13.6	104,220

	Bleeding Time, Min.	Coagulation Time, Min.
12/18	2	5
1/15	2	6

* 8 blast-like cells.
† 6 blast-like cells.
‡ 10 blast-like cells.

The blood urea nitrogen was 15 mg. per 100 cc.; alkaline phosphatase 3.3 units per 100 cc. The total proteins were 6.6 gm., with 4.3 gm. albumin and 2.3 gm. globulin, per 100 cc. A roentgenogram of the chest was normal.

Examination of the bone marrow on Feb. 6 showed 56% blast forms (lymphoblasts?).

The liver and spleen are not palpable, and there is no lymphadenopathy.

DISCUSSION

DR. WILBERT SACHS: I don't understand how the sternal puncture is consistent with leukemid in the face of the normal blood picture.

DR. HARRY SHATIN (by invitation): The bone marrow showed 56% blast forms. The hematologist assures us that the case was one of leukemia in an aleukemic form. It is of interest that the cutaneous manifestations appeared before the blood picture began to change. We first thought the eruption was a dermatitis medicamentosa, as it appeared 17 days after penicillin was administered. We are probably seeing the patient just at the beginning of his leukemia. The white cell and differential counts were normal on admission. We then found that the total white cell count was dropping but the relative lymphocytosis was increasing. We conceive of the possibility that the condition is a leukemid reaction due to a toxic condition. At present we are not going to treat the patient.

DR. WILBERT SACHS: I believe it is a drug eruption and that a biopsy should be performed. If the patient has leukemia, we should find the type of leukemia in the lesion. If we do, I think it is a drug eruption.

DR. ISADORE ROSEN: The patient's white cell count is as low as 2,600. How does a hematologist make a diagnosis of aleukemic leukemia?

DR. HERMAN SHARLIT: His polymorphonuclear leucocytes are decreasing all the time. How can this blood picture be differentiated from agranulocytosis? If we accept that blood picture, I would also accept the possibility of drug eruption.

DR. GEORGE M. LEWIS: The history of urticarial lesions on the hands and feet, and what we see of the remnants, are so typical of a penicillin rash that I think we must so diagnose the eruption. Leukemia may also be present.

DR. DAVID BLOOM: The patient received barbiturates, which might have produced this blood picture.

DR. ORLANDO CANIZARES: When we saw the patient, we did not know of the bone marrow report, but we considered the possibility of a blood change due to a drug eruption. Part of the picture may be due to barbiturates and part to penicillin, but I wonder whether anyone has seen

a blood picture similar to this from penicillin alone? I have seen it reported. The lesions on the hand and the swelling of the extremities are typical of a penicillin reaction, and it would be rather unusual to put a patient in the hospital for something else and have leukemia develop right before one's eyes.

NOTE: The patient was later discharged with a diagnosis of acute leukemia, probably lymphoblastic, and was admitted to another hospital a few days thereafter.

Case for Diagnosis: Pemphigoid Eruption? Presented by DR. HERMAN SHARLIT.

W. C., a man aged 57, is presented from the New York Skin and Cancer Hospital with an eruption of two months' duration. The lesions involved both axillas, the forearms somewhat, but especially the lower part of the abdomen, the groins, and genital area. They consist of erythema with some crusted (ruptured) bullae and some scaly, pea-sized to larger individual units. There is no history of drug ingestion. Treatment has thus far been ineffective.

The urine, blood cell count, erythrocyte sedimentation rate, and Wassermann reaction of the blood were not pathologic. A biopsy specimen from the groin was reported as showing contact eczematous dermatitis.

DISCUSSION

DR. DAVID BLOOM: The appearance of the crusted discrete lesions and the finding of a bulla in the epidermis on microscopic examination suggest a bullous eruption. Whether it is bullous impetigo or of the nature of pemphigus erythematosus cannot be decided now.

DR. PERRY SACHS: Microscopically, pemphigus must be seriously considered.

DR. GEORGE M. LEWIS: The clinical picture could fit in with pyogenic eczema, and I would favor that diagnosis. Seborrheic dermatitis is also a possibility.

Dermatofibrosarcoma Protuberans. Presented by DR. NATHAN SOBEL.

B. M., a man aged 37, was first seen at the New York Skin and Cancer Hospital on March 30, 1949. On the inner aspect of the upper part of the right thigh was a pedunculated, marble-sized, smooth fibroma of three months' duration. This was excised. On April 6, 1951, the patient returned because of a recurrence; at the same site there was an irregular plaque, measuring about 2.5 by 2 in. (7.5 by 5 cm.). The plaque was infiltrated and consisted of nodules and two hazelnut-sized tumors attached to the skin with broad pedicles. One of these had appeared only two to three weeks previously and had grown rapidly. Histologic examination of a pedunculated tumor showed it to be a fibroma with myxomatous changes—an infiltrated lesion fibroma. The entire area was excised on June 29, and histologic examination of the specimen showed a cellular fibroma.

On Aug. 13, a nodule ¼ in. (0.6 cm.) in diameter was noted in the scar, about one-third the distance from its lower end. This nodule has remained fairly stationary, and no others have appeared. There is no adenopathy of the regional lymph nodes. The tumor consultation service recommended wide excision with plastic repair, to be performed after several months' further observation.

DISCUSSION

DR. DAVID BLOOM: The recurrence of the lesion forces one to doubt that this is a plain fibroma with myxomatous changes. The diagnosis as presented is most probably correct.

DR. WILBERT SACHS: I would agree with the idea of waiting for a while. Because of the tremdous myxomatous degeneration, much of the lesion may disappear. Spindle-cell sarcoma is highly malignant, and one does not get a history of three years' duration. I am inclined to believe the lesion is not a sarcoma, but a recurrence of a benign lesion.

Case for Diagnosis: Tuberculosis Cutis? Leprosy? Sarcoid? Presented by DR. DAVID BLOOM.

C. C., a Chinese man aged 21, has had an eruption on the right cheek for the past six months. There are two quarter-sized, round lesions, each of which is made up of tiny erythematous nodules. The color of the lower lesion is reddish-brown.

Serologic tests for syphilis, a complete blood cell count, and urinalysis gave normal findings. The intradermal test gave a 1+ reaction to tuberculin in a dilution of 1:1,000,000 and a 2+ reaction to a 1:100,000 dilution. Scrapings from the lesion did not reveal any acid-fast bacilli. The biopsy report was "sarcoid reaction."

DISCUSSION

DR. WILBERT SACHS: The number of tubercles in the section would rule out tuberculosis verrucosa cutis. I thought the process was lupus vulgaris rather than sarcoid. The Ziehl-Neelsen carbolfuchsin stain gave negative results.

DR. ORLANDO CANIZARES: The lepromin test should also be done.

DR. PERRY SACHS: The microscopic picture was that of Boeck's sarcoid, but the numerous giant cells suggest lupus vulgaris. Giant cells are not a feature of leprosy.

DR. DAVID BLOOM: Clinically, the lesions, particularly the lower one, suggest lupus vulgaris. Because of the strongly positive tuberculin reaction, sarcoid can be excluded. Because the patient was an Oriental, leprosy was considered and, I believe, eliminated. One may assume that pathologically this lesion is a transition between sarcoid and lupus vulgaris.

DR. WILBERT SACHS: You can have a sarcoid reaction with a great deal of inflammatory reaction, or lupus vulgaris with little, and there comes a point where it is impossible to say which it is. However, giant cells would make it appear to be sarcoid rather than lupus vulgaris. Sometimes I have seen slides in which such cells were the only feature.

Pityriasis Rubra Pilaris (?) in a Three-Year Old Child. Presented by DR. NATHAN SOBEL.
Case for Diagnosis: Pemphigus Vulgaris (?) Presented by DR. ORLANDO CANIZARES.
Dystrophia Mediana Unguius Canaliformis. Presented by DR. DAVID BLOOM.
Diplopia Associated with Leprosy (Neural). Presented by DR. MAURICE J. COSTELLO.
Multiple Squamous-Cell Carcinoma. Presented by DR. ORLANDO CANIZARES.
Boeck's Sarcoid. Presented by DR. ORLANDO CANIZARES.

Samuel M. Peck, M.D., *President*
George M. Lewis, M.D., *Secretary*
March 11, 1952

Keratosis Pilaris (Ulerythema Ophryogenes). Presented by DR. PAUL GROSS.

W. R., a boy aged 7 years, has had an eruption on the face since early infancy. He was born with red birthmarks on the forehead and on the back of the neck, but these have disappeared. The eruption on the cheeks, however, has been progressing. The mother states that the patient's father, who is dead, had a ruddy complexion which somewhat resembled the skin condition of the boy.

The eruption, which is strictly confined to the eyebrows, consists of pinpoint-sized, reddish papules and some pitted scars, with corresponding loss of hair. On both cheeks, but only in the preauricular portion, are many discrete, whitish or pink keratotic papules and small, light-brown, pitted scars. On the extensor surface of the forearms there are a few discrete, brownish, follicular keratotic cones, some of which contain an atrophic lanugo hair.

DISCUSSION

DR. PAUL GROSS: Would thorium X therapy be of any benefit in this case?

DR. JACK WOLF: Keratosis pilaris is a self-limited process ending in follicular atrophy. There is at present no satisfactory method of treatment, nor is one likely to develop which will improve upon the altogether spontaneous satisfactory end-result. Thorium X may hasten the end-result, but only at the possible expense of cutaneous changes from ionizing radiation.

DR. MAURICE J. COSTELLO: Isn't it true that there is a certain low-grade infection in this type of lesion which is the cause of the ultimate atrophy? I have treated patients with this derivatives of 8-hydroxyquinoline). If the lesions are not severe, this treatment might be worth while.

DR. SAMUEL M. PECK: I have treated a few of these patients, but I also considered the disease as probably a low-grade inflammation. I did exactly what Dr. Costello did except that I also gave my patients sulfonamide drugs. One had considerable irritation with higher concentrations of quinolor® and was not affected by aureomycin, penicillin ointment, or oxytetracycline (terramycin®) ointment.

DR. DAVID BLOOM: This is considered a severe type of keratosis (lichen) pilaris. Being a follicular abnormality, I think trial with vitamin A therapy is justified. Although I have used it in several cases, I am not sure that it is very effective, but it is as good a remedy as anything which has been proposed tonight.

DR. SAMUEL M. PECK: I don't understand the disease process; no matter what you do you are going to have scars; so I think the patient should be talked into having a biopsy. Very often you find you change your diagnosis. I had a female patient, older than this boy, and I thought the best cosmetic result was with Lassar's peeling paste (zinc oxide paste with salicyclic acid).

DR. JACK WOLF: I have followed a number of patients for several years. I have peeled the lesions with strong peeling agents, cold mercury-vapor light, etc. There isn't a single remedy that will do any good except for a short time.

DR. WILBERT SACHS: I don't know anyone who has had enough experience with histological sections to speak with authority; therefore I asked for a biopsy so as to learn something about the process, at least as seen in this patient. There are contradictory ideas here. If the lesion is nevoid, I cannot understand a persistent erythema that goes on to atrophy. Otherwise the atrophy should be present at the beginning, although perhaps not visible.

DR. PERRY SACHS: In some of these cases the disease has been called lichen planopilaris. Any atrophy would depend upon the lesion of the follicles, rather than on loss of tissue.

DR. PAUL GROSS: I am inclined to agree with Dr. Wolf that the case is one of a nevoid anomaly and that therefore therapy is of doubtful value. As for Dr. Costello's suggestion, it is true that some cases may be complicated by folliculitis, but in the present case there is no evidence of infection. Dr. Sachs could tell us more about the degenerative changes in the corium which are responsible for the atrophy. That the condition is not confined to the follicle is borne out by the clinical picture, which always includes various degrees of erythema of the entire area involved. I am skeptical about vitamin A therapy, even in simple keratosis pilaris; but therapeutic trial with moderate doses is justified.

Adenoma Sebaceum Associated with Gingival Fibromas. Presented by DR. PAUL GROSS.

R. H., a youth aged 20, was first seen in August, 1948. At that time he had an eruption on the nose and on the adjoining parts of the cheeks and upper lip which consisted of many small nodules, varying in size from that of a small to that of a large pinhead. Some of these lesions were skin-colored; others bright red, due to the presence of telangiectases. In addition, there were many dilated capillaries of the normal skin in the areas involved. There was also a fleshy nodule with an elongated hyperkeratotic tip growing out from the base of the nail of each fourth toe. A biopsy specimen taken at that time revealed some atrophy of the surface epithelium and cutis, as well as numerous somewhat twisted hair follicles, with some keratosis of the follicular orifice, telangiectases in the dermis, and a few cells containing hemosiderin pigment. There was no inflammatory reaction.

The patient presented no neurologic abnormalities and apparently showed no impairment of intelligence. He is attending college and is a good student.

The eruption was treated from August, 1948, to February, 1949, by electrocoagulation of the nodules and cryotherapy for the telagniectases. The cosmetic result has been satisfactory and permanent.

For the past two years nodules have been developing on the gums.

DISCUSSION

DR. HERMAN SHARLIT: Do the gums bleed extraordinarily? (Answer: No) I should ignore the mouth unless it caused trouble and bleeding.

DR. PAUL GROSS: A combination of adenoma sebaceum with true Recklinghausen disease (neurofibromatosis) is rare. There are lesions on the trunk in tuberous sclerosis which resemble neurofibromas. Butterworth has described a variety of such lesions in a classic paper on the subject.

Follicular Nevus. Presented by DR. NATHAN SOBEL.

L. C., a girl aged 12, is presented from the New York Skin and Cancer Unit with an irregularly shaped, well-defined plaque on the outer aspect of the right arm, composed of pinhead-sized lesions which suggest papules or cysts. It has been present since infancy.

The biopsy report was that of follicular cyst.

DISCUSSION

DR. WILBERT SACHS: I should not call the lesion a follicular cyst or follicular nevus clinically. I think it is a unilateral nevus, and I should like to see more biopsy sections in order to determine the type of nevus. I should be very much surprised if it were a pure nevus follicularis.

DR. GEORGE M. LEWIS: If this lesion were on the scalp, one would think of a sebaceous nevus, very superficial in type. The color and dome-shaped appearance give that impression.

DR. JESSE A. TOLMACH (by invitation): Most of the nevoid conditions one meets are difficult to diagnose accurately on a clinical basis and require histologic confirmation of the clinical picture.

DR. DAVID BLOOM: A provisional diagnosis of connective tissue nevus was made originally in this case. However, the biopsy report obliges one to change the diagnosis to follicular nevus.

DR. WILBERT SACHS: Nevus comedonicus is frequently associated with sebaceous nevus.

DR. JACK WOLF: The yellowish color of the lesion suggested to me, as it did to Dr. Lewis, the diagnosis of sebaceous nevus. However, I believe that this is the type of nevus in which histologic examination from different parts will show differing nevoid pathology.

DR. PAUL GROSS: Despite the histopathologic findings, I should agree with the clinical diagnosis of connective tissue nevus. The follicular cyst found on biopsy could have been of secondary or concomitant development. Certainly, this is not a nevus comedonicus, although this follicular type of nevus can occur on any part of the body.

DR. NATHAN SOBEL: I also thought it was a connective tissue nevus when I first saw the child.

NOTE: A second biopsy showed a dilated follicle filled with horny material and adjacent to it a follicular cyst. Otherwise the epidermis was essentially normal.

Dermatofibroma (Dermatofibrosarcoma) Protuberans. Presented by DR. ANTHONY C. CIPOLLARO.

D. L., a woman aged 48, was first seen on Feb. 23, 1952. She states that 15 years ago she had a small lump on the abdomen excised. Soon after its removal many other small lumps appeared in the same general area. The lesion has gradually spread to its present size. The lesion which was removed was thought to be a cyst. There has been no other treatment. A biopsy in 1949 was reported as showing leiomyoma.

The patient now presents an elongated band-like lesion situated on the abdomen below the umbilicus, measuring approximately 5 by 1.5 in. (12.7 by 3.8 cm.). The individual nodules vary in color from pinkish to purplish, as well as in size. Lesions which cannot be seen can be palpated, especially at the periphery.

A biopsy, reported by Dr. Wilbert Sachs, showed a mass of spindle-shaped cells throughout the cutis. The collagen itself in places was edematous, compact, and arranged in bundles. The epidermis showed no significant changes. The diagnosis was fibroma.

DISCUSSION

DR. WILBERT SACHS: I agree with the clinical diagnosis, and the microscopic picture is that of dermatofibrosarcoma. The unfortunate part of the term is "sarcoma," and I think it refers only to its recurrence locally when removed, and not to any malignancy.

DR. MAURICE J. COSTELLO: I should certainly agree with the presenting diagnosis. It looks just like that in the case presented last month, although that lesion was smaller. With regard to the therapeutic approach, should it be widely excised? Do these lesions recur only when not completely excised? It is important to know that.

DR. SAMUEL M. PECK: My impression was that the best result one could get, even with wide excision, was a keloid.

DR. GEORGE M. LEWIS: This tumor is not invariably benign. There are instances of metastasis. For this reason, wide surgical excision is indicated.

DR. NATHAN SOBEL: In the case I presented last month the tumor recurred after fairly wide excision. We referred the patient to the tumor consultation service for opinion, and they felt the lesion could be sufficiently removed if widely and deeply excised. In that case the histopathology was also that of fibroma, characterized by constant recurrence. While the abdomen is the commonest site for these lesions, they can occur on the thigh, as in my case, or on other parts of the body.

DR. WILBERT SACHS: When the lesion is removed, it might be wise to do biopsies of peripheral areas.

DR. ANTHONY C. CIPOLLARO: I don't think it is proper to consider this lesion as entirely benign. Fibromas do not recur after removal. This is different from a fibroma, in spite of what we have seen microscopically; there is a tendency to keep on growing. We can't ignore the literature, and a sufficient number of cases have been reported in which there was a sarcomatous spread from lesions so diagnosed. I would feel uneasy about advising a woman of this age group to do nothing, and I think the tumor should be completely excised, widely and deeply. This woman has lesions where you can't see them. A whole inch beyond the visible border there are subcutaneous nodules. There should be extremely careful palpation before and even after excision. If we remove all of the lesion, we are likely to prevent recurrence at the local site. Whether or not there are cells of this type distant from the lesion only time can tell. No one knows the exact nature of this type of tumor; we don't know how they metastasize, and we don't know whether lesions similar to this develop elsewhere, in vital organs, as in Kaposi's sarcoma, in which they appear to develop de novo in practically every organ of the body. I am going to advise the patient to have the lesion excised.

Lymphogranuloma Venereum. Presented by DR. ANTHONY C. CIPOLLARO.

Lichen Planus Developing in an Atrophic Area? Presented by DR. PERRY M. SACHS.

MINNESOTA DERMATOLOGICAL SOCIETY

Elmer T. Ceder, M.D., *President*

Harold G. Ravits, M.D., *Secretary*

Feb. 16, 1952

Melanoma. Presented by DR. CARL W. LAYMON, Minneapolis.

Mrs. G. B., a white woman aged 82, had a brown patch appear on the right temporal region 12 years ago. The color had become darker in the past year. A rapidly growing nodule appeared in this area six months ago.

Examination shows a pigmented patch measuring 8 by 5 cm., with a bleeding, fungating, black central mass, 1.5 cm. in diameter. The lesion is located on the left temple.

Nevus Pigmentosus. Presented by DR. CARL W. LAYMON, Minneapolis.

D. L., a girl aged 3 months, was born with lesions on the head, trunk, and extremities.

Examination showed a large hairy verrucous nevus involving the right side of the scalp. Another patch was present on the left frontal region. Multiple smooth pigmented punctate and nummular lesions were present on the trunk, extremities, and left side of the scalp.

No therapy was instituted.

DISCUSSION

DR. S. ROTHMAN, Chicago: This melanoma arises from a flat, noninfiltrated, deep-brown pigmented spot. It would be interesting to know histologically whether this is lentigo maligna and whether the tumor arose after the flat pigmentation had been present for some time. Lentigo maligna is an intraepidermal melanoma, and lives can be saved if in this stage the lesion is broadly excised.

DR. STURE JOHNSON, Madison, Wis.: In several instances melanomas have been treated successfully at the Wisconsin General Hospital by chemosurgery (Mohs, F. E.: ARCH. DERMAT. & SYPH. **62**:269, 1950).

DR. H. E. MICHELSON, Minneapolis: I should like to ask Dr. Johnson what he means by fine results. Certainly the manner of removing a primary or visible lesion of malignant melanoma cannot be the important thing, providing it is as complete as a method allows. I doubt very much whether one method or another changes the prognosis.

DR. FRED BECKER, Duluth: I should like to raise the question of what is to be done with a child who has these extensive nevi at this early age. The apparently few nevi that undergo malignant change seem to warrant observation and removal when the child reaches full development rather than extensive surgical procedures at this age unless some change in the nevus occurs at this time.

DR. PAUL A. O'LEARY, Rochester: Our experience with melanomas in children differs from that of Pack. He is of the opinion that in children 15 years old or younger melanomas do not metastasize. Of 18 children we have seen with melanoma, 5 have died of metastatic lesions.

Large pigmented and hairy nevi may be removed surgically by excising strips of the nevus, suturing the edges, waiting until the wound heals, and repeating the process until the nevus has been entirely removed. The scar is not an ideal one, although cosmetically it is more attractive than the nevus.

DR. C. W. FINNERUD, Chicago: Some of these unfortunate patients with extensive pigmented nevi can be helped. Within the last week I saw a youth about 20 years old whom I first saw when he was a week old. At birth he had a pigmented, verrucous, hairy nevus of the left side of the face, diffusely involving the forehead, eyelids, nose, and upper left cheek. As the result of a series of skin grafts by Dr. G. B. New of the Mayo Clinic, such a remarkable cosmetic result has been accomplished, even with the eyelids, that no appreciable deformity is noticeable.

Case for Diagnosis: Features Resembling Pachydermoperiostosis. Presented by DR. H. E. MICHELSON, Minneapolis.

A white girl, now aged 7 years, had enlargement of the hands and feet and associated thickening and coarse wrinkling of the skin in these areas at approximately 2 to 3 years of age. It had an insidious onset and gradually increased in severity. During this time she exhibited hepatomegaly, steatorrhea, pseudohypoparathyroidism, and stunted growth. Teeth were carious, and episodes of hypocalcemic tetany occurred. Clinical improvement occurred with large oral doses of vitamin D and calcium, but no response to parathyroid hormone was noted. Throughout this period the skin continued to be dry and pruritic generally.

Examination reveals dry, thickened, coarsely wrinkled skin over the hands and feet, the condition fading out at the wrists and ankles. The face and scalp are not similarly involved. The skin generally is dry and pruritic.

Laboratory findings at present are normal except for a high fat content of the stool, the latter being increased in quantity. X-ray study of the skeletal structure of the hands and feet reveals broadening of the bones, with irregular trabeculation, and irregular calcification. A roentgenogram of the sella turcica is normal.

Histologic examination of a specimen from the wrist reveals hyperkeratosis and some elongation of rete pegs.

DISCUSSION

DR. JOHN D. KRAFCHUK, Minneapolis: The changes observed in the hands and feet of this young girl are similar to those described for pachydermoperiostosis, 60 reported cases of which were recently reviewed (Vague, J.: La pachydermopériostose, *Ann. med.* **52**:152, 1950). This

syndrome consists of (*a*) enlargement of the hands and feet; (*b*) thickening, with irreducible furrows and folds of the skin over the extremities, face, and scalp, and (*c*) periosteal proliferation, predominantly of the bones of the extremities. The skin changes are in many ways similar to those seen in acromegaly, while the roentgenologic findings resemble the picture of hypertrophic pneumonic (pulmonary) osteoarthropathy. However, characteristically, no pituitary tumor or chronic pulmonary disease can be demonstrated. Of the 60 reported cases, 56 involved males, with the usual time of onset shortly after puberty.

In the present case there is no laboratory or clinical evidence of acromegaly and, except for several transient episodes of pneumonitis in the past, no pulmonary disease. The chemical findings of parathyroid hormone deficiency were demonstrated, but no response to large doses of parathyroid hormone was noted. Calcium and phosphorus values became normal after prolonged oral administration of vitamin D and calcium. This case has been reported as one of pseudohypoparathyroidism (Lowe, C. E.; Ellinger, A. J., and Wright, W. S.: Pseudohypoparathyroidism: Seabright-Bantam Syndrome, *J. Pediat.* **36**:1, 1950). The present case is also unique, in its relation to pachydermoperiostosis, in its very early age of onset and its occurrence in a female.

Porokeratosis (Mibelli's Disease). Presented by DR. CARL W. LAYMON, Minneapolis.

T. P., a white boy aged 9 years, manifested a single, noninflammatory papule on the right thigh at the age of 6 months. The papule gradually enlarged peripherally and developed a thread-like border and an atrophic center. During the past two years several satellite lesions have appeared. There is no history of developmental anomalies in the patient's parents or siblings.

Examination shows a single, irregular circular, gray, noninflammatory plaque, measuring 2.5 by 5 cm., with a firm, thread-like encircling border and a depressed, atrophic center on the extensor surface of the right thigh. Small, milium-like bodies are embedded in the floor of the lesion. One large and several satellite lesions of similar characteristics are present.

The histologic findings are compatible with a diagnosis of porokeratosis.

DISCUSSION

DR. C. W. FINNERUD, Chicago: I have seen only two cases that everyone present agreed were unquestionable instances of porokeratosis, but I have seen at least a dozen in which there seemed to be just about as much to question then as there is in the present case. Although I could not detect the characteristic dyke grossly, the section did show a keratotic invagination of the epidermis in one area. The diagnosis as presented may be correct. At least, I cannot offer a better one.

DR. E. P. SCHOCH, Minneapolis: In a review article by Bloom and Abramowitz (ARCH. DERMAT. & SYPH. **47**:1, 1943) the authors pointed out that the name porokeratosis is a misnomer, since the hyperkeratosis characteristic of the disease is not limited to the pores. They concluded that the process was largely one of dyskeratosis, and not of a true nevus. The term keratodyskeratotic anomaly was preferred.

Jones and Smith (ARCH. DERMAT. & SYPH. **56**:425, 1947) have elaborated on the role of vitamin A deficiency as an etiologic possibility, but the case they reported failed to respond to vitamin A therapy. Similar experience was noted recently by Haber and Porter (*Brit. J. Dermat.* **63**:28, 1950).

The disease is considered a rarity, though over 150 cases have been reported to date.

Case for Diagnosis: Reticulohistiocytoma. Presented by DR. CARL W. LAYMON, Minneapolis.

In Mr. H. F., a white man aged 52, lesions appeared on the hands, on the bridge of the nose, and behind the ears about two months ago. A surgical procedure on the bowel was performed in 1951.

Examination shows firm, red nodules along the sides of the fingers, in the periungual regions, on the dorsal surfaces of the hands, on the bridge of the nose, and on the rim of the left ear.

DISCUSSION

DR. S. ROTHMAN, Chicago: I thought these lesions were carcinomatous metastases.

DR. R. W. GOLTZ, Minneapolis: When I looked at the histologic section, I considered the diagnosis of reticulohistiocytoma. The large foamy cells, many of them multinucleated, throughout the corium are identical with those in the two cases of this condition which we had the opportunity to study last year. Similar cases have been reported by Zak (*Brit. J. Dermat.* **62**:351, 1950) and by Caro and Senear at the American Dermatological Association meeting in June, 1951. I understand that special stains have not been made on this section. If the condition is reticulohistiocytoma, I would expect the stains for fat to be positive.

In this connection, I should like to mention terminology. This condition is, apparently, a newly recognized one, and I am not sure that the terms which have been used to describe it are the best possible. In the present case there was an explosive onset, with multiple lesions, and it would seem that the suffix "osis," rather than "oma," would be appropriate. Furthermore, in our previous cases the lesion did not show a definite relation to reticulin, as in Zak's case. Therefore, some such term as "histiocytosis cutis" might be more accurate. It would be interesting to see whether the same process is going on elsewhere in the reticuloendothelial system, and biopsies of the liver and lymph nodes might be considered.

DR. JOHN I. COE, Minneapolis: I feel that this lesion is not compatible with a metastatic carcinoma, at least none that I have ever seen. The cells resemble histiocytes, and I believe that this should be classified as some type of histiocytoma, the name being used as a general term. I should not wish to go further without being able to study the lesion more thoroughly.

DR. A. L. WELSH, Cincinnati: I believe this patient has reticulohistiocytoma cutis, both clinically and histologically.

We have had a similar case, with lesions distributed as in this case and, in addition, lesions on the palms and soles. The linear arrangement of the nodules along the sides of the fingers in symmetrical fashion rules against (clinically) any type of metastatic lesions. The nodules on the fingers resemble closely those in our case, and the pictures of reticulohistiocytoma cutis shown by Dr. Marcus Caro at the 1951 meeting of the American Dermatological Association.

DR. FRED BECKER, Duluth: I think this patient's lesions resemble very much those in the case I presented at the meeting of this Society in Duluth in 1950. Lesions are beginning to develop along the ear, and the histologic appearance is very similar. Dr. Caro will soon publish a report of his case, probably with the diagnosis of histiocytoma.

DR. CARL W. LAYMON, Minneapolis: A supraclavicular node developed which was thought by the surgeons to be metastatic. Before the patient was presented, we had an opportunity to see him only once. For this reason, we thought it was best to present him for diagnosis.

DR. J. F. MADDEN, St. Paul: I do not think that you can rule out metastatic carcinoma because of the highly malignant carcinoma removed from the intestine and the metastatic supraclavicular node that was found.

Lichen Sclerosus et Atrophicus. Presented by DR. CARL W. LAYMON, Minneapolis.
Sarcoidosis Associated with Lichen Sclerosus et Atrophicus. Presented by DR. ELMER T. CEDER, Minneapolis.
Sarcoidosis. Presented by DR. CARL W. LAYMON, Minneapolis.
Pityriasis Rubra Pilaris. Presented by DR. CARL W. LAYMON, Minneapolis.
Case for Diagnosis: Steatocystoma Multiplex or Syringocystoma? Presented by DR. CARL W. LAYMON, Minneapolis.
Neurofibroma. Presented by DR. CARL W. LAYMON, Minneapolis.
Subacute Lupus Erythematosus. Presented by DR. CARL W. LAYMON, Minneapolis.
Acute Systemic Lupus Erythematosus ("Cured?"). Presented by DR. CARL W. LAYMON, Minneapolis.
Urticaria Pigmentosa. Presented by DR. CARL W. LAYMON, Minneapolis.
Pigmented Purpuric Lichenoid Dermatitis. Presented by DR. CARL W. LAYMON, Minneapolis.
Calcinosis Cutis. Presented by DR. CARL W. LAYMON, Minneapolis.
Lipomelanotic Reticulosis. Presented by DR. CARL W. LAYMON, Minneapolis.
Prurigo Nodularis. Presented by DR. CARL W. LAYMON, Minneapolis.
"Pat and Mike" Case for Diagnosis. Presented by DR. CARL W. LAYMON, Minneapolis.
Nevus Verrucosus. Presented by DR. CARL W. LAYMON, Minneapolis.
Nevus Unius Lateris. Presented by DR. CARL W. LAYMON, Minneapolis.
Keratosis Palmaris et Plantaris. Presented by DR. CARL W. LAYMON, Minneapolis.
Keratoderma Punctata. Presented by DR. CARL W. LAYMON, Minneapolis.
Arsenical Keratosis. Presented by DR. ELMER M. RUSTEN, Minneapolis.

NEW YORK DERMATOLOGICAL SOCIETY

Anthony C. Cipollaro, M.D., *President*

Maurice J. Costello, M.D., *Secretary*

Jan. 22, 1952

Case for Therapeutic Suggestions: Chronic Lupus Erythematosus; Impending Dissemination? Presented by DR. FRANK E. CORMIA.

E. S., a woman aged 36, has always enjoyed fairly good general health except for the present trouble. Her periods began at the age of 14 and were normal until 10 years ago, since which time they have lasted only one day. Seven years ago four abscessed teeth were found, and because of her skin eruption all the teeth were removed.

Lesions involving a small area on each cheek appeared at the age of 14 with the onset of her first menstrual period, and a few weeks after her father committed suicide. After therapy with some sort of lamp the eruption spread widely over the face. Lesions appeared on the fingers and hands four years ago. During the past few weeks ill-defined erythematous lesions have developed on the lower extremities. Sunlight has a variable effect on the eruption, but menstrual flare-ups are the rule. At the age of 17 a severe secondary infection required hospitalization for 2 weeks. Secondary infection again supervened in 1947 and again two weeks ago. One week ago a localized cellulitis was noted in the right leg.

The patient has been treated for many years with more than 100 injections of bismuth, many injections of bismuth arsphenamine (bismarsen®) and gold, and 30 injections of suramin (naphuride®) sodium. Quinine, Asiatic pills (pills containing arsenous oxide and black pepper), and "bistrimate" (bismuth sodiumtriglycollamate) tablets have been given. Penicillin was administered daily for a month in 1947, with no effect except on an incidental secondary infection. She has received no injections in the last three years, but took a considerable amount of vitamin A last summer. She has never noted improvement from treatment. A pronounced personality change has followed the many years of partial disablement. The diet is high in carbohydrates and deficient in proteins and vitamins. Penicillin intramuscularly and bacitracin ointment were given for the recent infectious complication.

Chronic atrophic and erythematous patches of chronic lupus erythematosus are present on the scalp, face, chest, and hands. A subsiding secondary infection may still be seen, and mild cellulitis is present on the right leg. Mild erythematopapular eruptions and a few purpuric lesions are present on the lower extremities.

A study of the peripheral blood for L. E. cells was negative, Biopsy has not been performed, and blood counts, determination of the erythrocyte sedimentation rate, and other tests have been deferred because of the acute infection.

Because of the chronic disablement and progressive eruption, the patient is presented for therapeutic suggestion.

DISCUSSION

DR. GEORGE M. LEWIS: I am somewhat dubious about the prognosis. The lesions individually look like discoid lupus erythematosus, and atrophy is present, but this doesn't necessarily rule out systemic involvement. She should be treated very carefully for fear of inducing dissemination. I favor liver injections and the avoidance of bismuth, gold, and other conventional types of therapy for discoid lupus erythematosus.

DR. HARVEY BLANK (by invitation), Philadelphia: Page, in an important article on the use of quinacrine (atabrine®) hydrochloride U. S. P., in chronic discoid and subacute lupus erythematosus (*Lancet* **2**:755 [Oct. 27]1951), reports 18 cases, with astonishingly good results in 17, including those of the subacute disseminated form. Even the arthritic symptoms were greatly improved. Page felt that orally administered quinacrine hydrochloride acted as a light screen, and he demonstrated an increased tolerance to ultraviolet radiation following its use. The authors stated that good effects were not achieved until there was some yellow staining of the skin. The drug was given in doses of 100 to 300 mg. a day.

DR. JAMES M. FLOOD (by invitation), Sayre, Pa.: There are a large number of pustules on her hands, and I think you might find hemolytic streptococci there. I am wondering whether

these lesions are not a complicating feature. She also has marked flare-ups at the time of her menses. If we can be sure she is showing signs of dissemination, I think castration should be considered, although it is a serious measure and the decision should be carefully weighed.

DR. ANTHONY C. CIPOLLARO: I believe this woman now has acute disseminated lupus erythematosus. I think this case is a rare example of disseminated discoid lupus erythematosus developing features of an erythema-multiforme-like eruption and then going into the acute disseminated phase. I agree with those who consider this condition to be very serious, with a poor prognosis, and in view of that I recommend treatment with cortisone or corticotropin (ACTH) in preference to castration at present. I would prefer to begin with more conservative methods. Castration probably has been helpful in some cases, but since it is a serious surgical procedure, other therapeutic methods of proved value in some of these severe cases should first be employed.

DR. FRANK E. CORMIA: I was interested in Dr. Cipollaro's remarks and should like to ask whether he feels there is any danger in using corticotropin or cortisone. If there is impending dissemination, may we not do more harm than good with the corticosteroids?

DR. ANTHONY C. CIPOLLARO: Yes, it is dangerous; but we are dealing with a disease that may terminate fatally unless drastic measures are taken, and I think we are justified in using drugs as potent as the corticosteroids.

NOTE: This patient was presented at the meeting of the New York Dermatological Society on May 27, 1952, after castration.

Lupus Erythematosus: Subacute Residual Activity. Presented by DR. FRANK E. CORMIA.

M. P., a woman aged 26, had erythema of the hands following a severe sunburn 3½ years ago. Discoid lupus erythematosus in typical butterfly distribution developed in a few months. Three years ago fatigue and weakness were noted, and during the following year fever and pains in the chest and joints developed. She became acutely ill 14 months ago and was hospitalized in Sayre, Pa., under the care of Dr. James Flood. Manifestations at that time were old butterfly lesions, an acute lesion of the upper lip, and generalized acute arthritis. There were diplopia due to paralysis of the right rectus muscle, elevation in temperature to 103 F., a pulse rate of 130 per minute, 2,800,000 red blood cells and 8,500 white blood cells per cubic millimeter, and urinary casts. The sternal marrow examination was negative for L. E. cells.

Treatment consisted of six transfusions, intramuscular administration of corticotropin, 60 mg., decreased to 10 mg., daily over a period of six weeks, and surgical castration. All symptoms disappeared except for residual stiffness in the knee joints.

The patient has been followed at the New York Hospital for the past eight months. Chest pains were formerly present, but roentgenograms of the lungs revealed nothing abnormal. Recurrent swelling of joints has been noted. The skin has remained clear. Three months ago an intercurrent gastric ulcer developed but has now subsided. At present she feels fairly well except for easy fatigability, occasional depression, and a 30 lb. (13.6 kg.) gain in weight. No active cutaneous lesions are present, but there is some swelling of the knees.

The erythrocyte sedimentation rate has ranged from 26 to 33 mm. per hour. The complete blood cell count was normal. Total proteins were 8 gm. per 100 cc., with 4.3 gm. of albumin and 3.7 gm. of globulin. The cholesterol was 212 mg. per 100 cc. A kidney function test and an electrocardiogram revealed a normal condition. L. E. cells were found in the sternal marrow three months ago, but were not found in blister fluid one month ago.

Treatment has been symptomatic, and directed toward relief of the arthritis and the ulcer. Vitamin E (tofaxin®), in addition, has been given, 300 gm. daily by mouth and 100 mg. by muscular injection.

DISCUSSION

DR. GEORGE M. LEWIS: It seems like a good result. The condition is at least in a remission. The fact that she is up and around is evidence of that.

DR. JAMES FLOOD (by invitation), Sayre, Pa.: The patient was extremely ill when first seen, with a temperature of 104 F., great loss of weight, severe anemia, and polyserositis. She responded to corticotropin, but her symptoms recurred when the dose was lowered. Her financial circumstances were such that she would not have been able to continue treatment with cortisone;

so castration was suggested. The pros and cons were discussed; she had been married and divorced and had had one child. All in all it was thought the best thing to do. I am sure she would not have been alive had it not been for corticotropin. I think the fact that improvement has been maintained although she has had no cortisone or corticotropin since last year is indicative of a remission. I feel she would have had a relapse without castration.

DR. FRANK E. CORMIA: This woman is still suffering from lupus erythematosus. She has occasional arthritis; L. E. cells were found a few months ago, and there is evidence of the disease in a low-grade form. However, from the past history it is amazing that she is here at all, and I certainly congratulate Dr. Flood.

Leukoplakia. Presented by DR. FRANK E. CORMIA.

A. K. is a generally healthy woman aged 43. Her mother died of carcinoma of the rectum at the age of 54. The patient was first seen six months ago with a discrete white patch, clinically typical of leukoplakia, on the right side of the tongue. She had been seen by an otolaryngologist one month previously, a biopsy performed, a diagnosis of papilloma made, and no treatment recommended. When first seen by the presenter, the lesion was removed by electrocoagulation. Three and a half months later new whitish areas had developed around the coagulated area, and some burning and tenderness were present. The question was then raised whether possible irritation from "ammident" tooth paste and from a jagged tooth edge was present. These possible factors were corrected, and temporary improvement ensued. However, the process was again worse two weeks ago, and a second biopsy was performed. At this time therapy with crude liver extract and vitamin B was initiated.

On the right side of the tongue is a pecan-sized central area of irritable-looking mucous membranes. Surrounding this is a variable, whitish border.

A serologic test for syphilis was negative.

The biopsy of June 29, 1951, was reported as showing a small papillary growth, the surface of which was covered with stratified squamous epithelium thrown into folds. The basal layer was everywhere intact. It was supported by a small amount of richly vascularized connective tissue sparsely infiltrated with chronic inflammatory cells. The diagnosis was papilloma.

The biopsy of Nov. 29, 1951, reported by Dr. Wilbert Sachs, showed the epidermis to be tremendously and irregularly acanthotic. There was no granular or horny layer, and the palisade layer was intact. Although the tissue was somewhat disorganized, there was an increase in the number of blood vessels in the subepidermic region. There were also some fibrosis and a moderate infiltration of small round and wandering connective tissue cells. There were some polymorphonuclear cells. No evidence of lichen planus was seen. The diagnosis was probable leukoplakia with no malignancy.

DISCUSSION

DR. L. P. BARKER: All sources of possible irritation in the mouth should be eliminated. She should stop smoking entirely, even though she now claims to smoke only an occasional cigarette. There is an adjacent gold and silver filling in the teeth opposite the area of leukoplakia. Because of the possibility of irritation from the current that may be produced by dissimilar metals, this condition should be corrected.

I think the lesions should be destroyed as soon as all sources of irritation have been eliminated. I have seen no beneficial results in leukoplakia from the administration of vitamin B complex, or from the use of vitamin A, as suggested by some gynecologists. They use 200,000 U. S. P. units of vitamin A daily by mouth and 100,000 U. S. P. units three times a week by injection for leukoplakia of the vulva.

DR. GEORGE M. LEWIS: An occasional cigarette is enough to keep the process going. This is one condition in which the patient must abstain completely from tobacco in any form.

DR. ANTHONY C. CIPOLLARO: Two of the members spoke of yeast and liver injections. That method of treatment was popular several years ago, and evidence of its efficacy in the treatment of leukoplakia is lacking. I agree that the patches ought to be watched. If the patient is sufficiently intelligent to be under the observation of an expert, I think there is no danger. If changes do take place, it won't be overnight. It takes months, and perhaps years, before malignancy develops. I agree this woman has leukoplakia now, and my method of treatment is to keep her under observation.

DR. FRANK E. CORMIA: This patient has been quite a problem. When I first saw her, she had an elevated, localized plaque, about Grade 2+ to 3—. I thought I had electrocoagulated it all, but may have missed some of it. What amazed me was the rapidity with which it recurred. The type of leukoplakia now present is quite different from that I first saw.

Case for Diagnosis: Subacute Disseminated Lupus Erythematosus? Dermatomyositis?

Presented by DR. GEORGE M. LEWIS.

I. F., a woman aged 48, is presented from the New York Hospital with an eruption of the face, the V of the neck, and the fingers, of six months' duration. In mid-July, 1951, an erythematous "lumpy" eruption appeared on the palmar aspects of the fingers and erythema on the V of the neck. The patient had for several weeks taken a reducing medicine containing amphetamine, phenobarbital, and aloin; this was discontinued, but the eruption remained and became aggravated on the neck after exposure to sunlight. She consulted a dermatologist, who prescribed "green pills," an ointment, and cold compresses and told her she probably had either a drug rash or sensitivity to a detergent. The eruption subsided somewhat. In mid-August her fingers became swollen, stiff, and difficult to move. Within a short time most of the joints of the body became painful and stiff, with tenderness of the long bones. Erythematous "lumps" appeared on the elbows, scalp, and forehead. There was no tenderness of the muscles. Periorbital edema developed, with a blotchy, erythematous, macular rash on the midportion of the face. Exposure to sun or wind increased the erythema and caused burning.

The patient was given cortisone by mouth, 25 mg. every 4 hours for 10 days and then tapering-off doses for the next 10 days. During this time there were a loss of 12 lb. (5.4 kg.) in weight, fatigue, and loss of appetite. After cortisone therapy there was some improvement, with disappearance of the lesions on the forehead, scalp, and elbows. On Sept. 13 she was admitted to the medical service for study and further treatment.

Examination showed periorbital edema of the "butterfly" area of the face and the V of the neck, and edematous, erythematous skin over the distal phalanges. There was a retinal hemorrhage of the right eye. The shoulder, wrist, knee, and left hip joints were tender and painful, but not swollen. There was a weak grip bilaterally, but strength was otherwise good. No muscle tenderness was elicited.

The urine was found to be normal on repeated examinations. Complete blood cell counts showed 3,600 white cells per cubic millimeter on admission, but were otherwise normal. The erythrocyte sedimentation rate was 52 mm. per hour. The albumin-globulin ratio was 3.8: 3.1. The peripheral blood was negative for L. E. cells. Roentgenograms of the chest and hands revealed nothing abnormal.

From Sept. 13 to Oct. 23 the patient was treated with corticotropin, intravenously or intramuscularly, beginning with 40 mg. a day and tapering off to 15 mg. a day. During this time the patient's arthritic symptoms disappeared and the erythema decreased. She was taught to inject the corticotropin herself and was discharged on Oct. 23 and instructed to take 15 mg. twice daily, which was later increased to three times daily. In November this was changed to corticotropin gel, 40 mg. daily. After this the hands became redder and the fingers harder. In December the therapy was changed to oral use of cortisone, 25 mg. twice daily, which the patient is still taking.

The patient was referred to the department of dermatology for evaluation of a painful nodule of the right ear. This appeared to be chondrodermatitis nodularis chronica helicis, and the lesion was desiccated and curetted.

At present, the white blood cell count is in the neighborhood of 9,000 per cubic millimeter, and the erythrocyte sedimentation rate is about 30 mm. per hour. The albumin-globulin ratio has tended to return to normal.

DISCUSSION

DR. FRANK E. CORMIA: This patient was presented at our hospital conference last week. We weighed the pros and cons, and there was a difference of opinion. However, I did feel that the condition was probably dermatomyositis rather than lupus erythematosus.

DR. JAMES FLOOD (by invitation), Sayre, Pa.: It is a remote possibility, but as I looked at the patient's hand, I couldn't help but think she might have an erysipeloid infection. With this

there is very little pain, but marked tenderness and the dusky-red color are characteristic. There are chronic forms in which the patient runs a low-grade fever, with toxic effects.

DR. GEORGE M. LEWIS: In a negative way, the absence of red blood cells in the urine is important, and the fact that leucopenia is no longer present—and spontaneously disappeared—would tend to speak against acute disseminated lupus erythmatosus. Also, the patient is out of the usual age range. It is of interest to me that the consensus seems to favor dermatomyositis. Of course, the diseases do merge into one another. Another feature, which is probably nonspecific but worth calling attention to, is the diffuse alopecia. Periarteritis nodosa has not been entirely ruled out as a possibility.

Radiodermatitis of the Face Successfully Treated by a Split-Thickness Graft from the Abdomen. Presented by DR. MAURICE J. COSTELLO.

M. B., a woman aged 52, whose case was previously presented before this society as that of radiodermatitis complicated by painful keratoses, was operated on July 17, 1951. A split-thickness graft was removed from the lower part of the abdomen and sutured to the prepared area of the face, including the lower mandibular area. The operation has been followed by a good therapeutic and cosmetic result.

Vesicular Trichophyton Rubrum (Purpureum) Infection Simulating Dermatitis Herpetiformis. Presented by DR. MAURICE J. COSTELLO.

A. R., an Italian housewife aged 48, has had a generalized, extremely pruritic vesiculobullous, roughly symmetrical eruption of the extremities and torso for the past 15 years. Because of the symmetry, generalization, extreme pruritus, grouping, and the papulovesicular nature of the eruption, the diagnosis of dermatitis herpetiformis was made; and because the patient improved under sulfapyridine therapy, there was no reason to change the diagnosis. Recently because of the arciform appearance of the lesions in the axilla and pubic region, a culture was made of the vesicular fluid and a diagnosis of Trichophyton rubrum infection was made from the characteristics of the culture growth. The patient has had indifferent treatment because the presenter did not wish to change the clinical characteristics of the present fungous infection before presentation.

DISCUSSION

DR. GEORGE M. LEWIS: The tip-off, clinically, is the continuity of the plaque; the lesions are not entirely discrete, but they are all contained within the border of a scalloped and serpiginous area. The symmetry of the lesions is interesting and would make one think very much of dermatitis herpetiformis. In the disseminated forms symmetry is more commonly noted, whereas in the more limited areas one will find unilateral involvement. For instance, tinea cruris due to T. rubrum is almost always unilateral, whereas with the Epidermophyton disease a symmetrical bilateral eruption is characteristic. It is also common to see one hand affected, rather than both. The lesson is that we should study cultures more than we do in cases of recalcitrant lesions.

DR. L. P. BARKER: I agree with Dr. Lewis that it is important to take cultures in all cases of scaly dermatoses in which the diagnosis is in question.

DR. HARVEY BLANK (by invitation), Philadelphia: We observed several such cases in India during the war, in which the T. rubrum infection extended over the body, including the face. The lesions would disappear spontaneously when the man was sent to a cool "hill station," but in hot jungle regions they were practically impossible to cure.

DR. PAUL E. BECHET, Elizabeth, N. J.: It is interesting to note the marked similarity of the eruption to dermatitis herpetiformis, but the diagnosis has been proved culturally; so it is definitely a fungous infection. A clinical point in favor of dermatophytosis is the location in the axillas, crotch, intergluteal folds, etc. I should like to suggest a remedy which rarely sets up irritation, despite its high content of sulfur. It consists of sulfur, 4 dr. (15 gm.); olive oil, 3 dr. (11.25 gm); white petrolatum ad 1 oz. (30 gm.). The lack of irritation depends on how it is mixed. The oil and sulfur are mixed thoroughly before the petrolatum is added. Why a 10% sulfur salve will not infrequently set up a dermatitis and a properly mixed 40% one will not produce irritations is not accurately known.

DR. ANTHONY C. CIPOLLARO: This woman with an extensive T. rubrum infection did not have toe nail involvement. Every patient with T. rubrum infection that I have seen has always shown the moccasin type of eruption, to which Dr. Lewis first called my attention. I always look for that, no matter where the lesions are on the body.

DR. GEORGE M. LEWIS: There is no rule as to prognosis in these cases. However, the outlook is somewhat more favorable in cases of this type than in some of the more localized eruptions. One of my favorite drugs in the treatment of this infection is chrysarobin. In such a widespread, acute inflammatory expression it would necessarily be used with caution.

Psoriasiform Erythroderma. Presented by DR. FRANK E. CORMIA.

P. M., a man aged 55, has had an increasingly severe psoriasis of the plaque type for 20 years. There were temporary improvements and remissions following low-dosage, intermittent cortisone therapy for a four-month period last year. Conventional treatment over the years has been of very little value. Two weeks in Florida, four years ago, resulted in temporary involution of lesions. In December, 1951, he took 200 mg. of cortisone for three days. Two days later, a generalized psoriasiform erythroderma supervened.

Two years ago mild rheumatoid arthritis involving the ankles developed. This improved greatly after seven weeks' treatment with colloidal gold and colloidal iodine. He has had minimal arthritic symptoms since that time.

There is generalized, somewhat infiltrated, erythematous and scaly psoriasis over the entire body. The patient is presented to illustrate the development of psoriasiform erythroderma after sudden withdrawal of cortisone given in full dosage.

DISCUSSION

DR. PAUL E. BECHET, Elizabeth, N. J.: I do not believe that psoriasis per se will develop into exfoliative dermatitis without some trigger mechanism, such as the use of concentrated ointments or sensitivity to a particular ingredient in the ointment.

DR. GEORGE M. LEWIS: This appears to be a most unfavorable effect of cortisone, and one that we should point out to general practitioners and others who are giving cortisone ad lib.

DR. JAMES FLOOD (by invitation), Sayre, Pa.: This patient has had cortisone for some time, and it might be that the adrenal cortex is now becoming a little atrophied. Perhaps corticotropin would stimulate the adrenal cortex, and be a better drug to use if it becomes necessary to use cortisone or corticotropin.

DR. ANTHONY C. CIPOLLARO: We saw these patients before we had corticotropin or cortisone. Perhaps the sensitivity is just a trigger mechanism, as Dr. Bechet mentioned.

DR. FRANK E. CORMIA: This patient had no local treatment whatsoever. He gave himself cortisone for three days, and two days later the erythroderma ensued. I present him to show that mild psoriasis may be seriously aggravated by the corticosteroids. We should be extremely cautious about the use of cortisone in this disease.

Dermatitis Herpetiformis Improved with Promacetin® Therapy. Presented by DR. MAURICE J. COSTELLO.

Vesicular Eczema from Foods. Presented by DR. FRANK E. CORMIA.

Anthony C. Cipollaro, M.D., *President*

Maurice J. Costello, M.D., *Secretary*

Feb. 26, 1952

Lichen Ruber Moniliformis. Presented by DR. PAUL E. BECHET, Elizabeth, N. J.

V. D., a woman aged 43, was first seen on July 11, 1951, with a dermatosis of six months' duration. A generalized, thickened, red eruption covered the entire face and neck. There was complete loss of the hair of the eyebrows and lids. Pruritus was intense. The trunk and legs were free of involvement, but both arms presented a curious clinical picture; the extensor surfaces showed the same type of eruption as the face, but the flexor surfaces exhibited long red striae, consisting of nodules in bead-like formation, with small, flattish, punctiform, shiny, non-inflammatory papules between the striae.

The blood cholesterol and hemogram were normal. The histologic picture was reported by Dr. Wilbert Sachs as inconclusive.

The lesions on the arm exactly resembled those of Wise and Rein's patient (ARCH. DERMAT. & SYPH. **35**:830, 1936). As in their patient, the microscopic picture did not suggest lichen planus, and Wise, therefore, preferred the designation of morbus moniliformis lichenoides.

DISCUSSION

DR. WILBERT SACHS: Many of us remember well the case of Wise and Rein. Of course, the papules stood out much more clearly than those we see in this patient. Neither did the patient have anything resembling a poikiloderma effect. I would not have thought of that diagnosis clinically and microscopically I could not make it. I did find some dilated, thickened vessels; edema, and considerable basophilic degeneration; I thought there was a possibility that the degenerative changes might have followed radiation therapy. I saw no cellular reaction at all, but possibly the cortisone removed this. There was no poikiloderma and no lichen planus. If it is true that the basophilic degeneration is due to x-irradiation and to the lack of cellular reaction to cortisone, the process may be some type of neurodermatitic reaction.

DR. C. T. CHIARAMONTE (by invitation), Brooklyn: I am inclined to agree with the presenter. When the skin is stretched, individual elements in linear formation become apparent. Because of involution, the papules are smaller than usual.

DR. FRANK E. CORMIA: The ichthyosis and keratosis pilaris are apparently incidental to the bizarre eruption on the upper extremities. First, I should like to ask whether there is a characteristic lichen planus picture histologically in lichen ruber moniliformis, and, second, whether there is any information that the treatment for lichen planus will cure or improve this condition.

DR. WILBERT SACHS: I should like to ask the presenter whether the patient had sufficient x-ray therapy to cause some of these changes. There are features in the slide to show that there may have been considerable exposure to x-rays. (Answer: The history states that she had two treatments, 75 r each.) She has entirely too much basophilic degeneration; I should have thought she had had much more therapy.

DR. GEORGE M. LEWIS: There is not sufficient evidence at this time to indicate a diagnosis of lichen ruber moniliformis. Assuming that the diagnosis is correct as presented, this case is probably the first in which cortisone therapy was used, and the result so far is excellent.

DR. EUGENE F. TRAUB: We don't know how much change there has been, but from the picture this evening no one could confirm the presenter's diagnosis. As of this evening, the patient presents keratosis pilaris and something more than mild xeroderma or ichthyosis. Dr. Costello's suggestion that some kind of ectodermal defect is involved would seem best to cover all angles. The eruption may have changed entirely as a result of the administration of cortisone.

DR. GERALD F. MACHACEK: Isn't the eruption a little too red for simple ichthyosis? She has a diffuse, macular erythema.

DR. EUGENE F. TRAUB: I believe the patient undoubtedly had a superimposed dermatitis or eczematization that has now practically cleared up, leaving xeroderma or ichthyosis. People with an extremely oily or dry skin are more liable to eczematization.

DR. PAUL E. BECHET, Elizabeth, N. J.: It is taking the members of the Society somewhat at a disadvantage to exhibit the patient with the clinical picture she presents tonight. If you had seen her two months ago, you would unanimously have agreed with my diagnosis, for she presented the characteristic picture on her arms. As a few doses of x-rays proved only slightly beneficial, this therapy was discontinued. In view of the usual negative therapeutic response to this disease, the patient was given 25 mg. of cortisone (cortone®) acetate by mouth three times a day. Improvement was dramatic, with no untoward results. The eruption has lost most of its characteristic appearance, so that I can easily understand the difficulty of making a diagnosis at present.

DR. WILBERT SACHS: In lichen ruber moniliformis the picture is like that of lichen planus because it is a variety of lichen planus. However, if it is morbus moniliformis, it is not lichen planus. In Wise's case one did not find lichen planus.

DR. PAUL E. BECHET, Elizabeth, N. J.: In Wise and Rein's case the histologic picture was not that of lichen planus. The elder Unna stated that the histologic picture in Kaposi's original case was not that of lichen planus. Because of these and two other cases, Wise and Rein proposed the name morbus moniliformis lichenoides, so as to avoid any confusion with lichen planus.

Diplopia Associated with Leprosy. Presented by DR. MAURICE J. COSTELLO.

J. L., an Italian barber aged 60, has been presented before this and other dermatological societies. He is presented tonight because of double vision which occurs when he turns his head to the right. Neuritic pains have been persistent and resistant to all forms of therapy, including promacetin® (sodium 4,4'-diaminodiphenylsulfone-2-N-acetylsulfonamide).

Pemphigus Vulgaris with Permanent Alopecia and Atrophy of Scalp: Cutaneous Lesion Improved with Neomycin Ointment. Presented by DR. MAURICE J. COSTELLO.

F. G., a man aged 60, was treated at Bellevue Hospital five years ago for syphilis of the central nervous system. He also had what was diagnosed at that time as pemphigus vulgaris. He has been previously presented before this Society because of remission of the disease and because of the permanent loss of hair with atrophy of the scalp. The alopecia has spread in peripheral fashion only. The patient always has had a number of crusted lesions on the periphery of the involved area. Prior to the use of neomycin ointment he had received aureomycin ointment, streptomycin ointment, and chloramphenicol (chloromycetin®) ointment, and these drugs were also administered orally. He received cortisone, without benefit. The remission of his generalized pemphigus five years ago was thought to have been brought about by carbarsone.

DISCUSSION

DR. WILBERT SACHS: I do not recall ever having seen atrophic lesions in pemphigus.

DR. FRANK E. CORMIA: We must congratulate Dr. Costello on a good result. Apparently, this type of secondary infection is particularly amenable to treatment with neomycin. I assume Dr. Costello will discuss the fact that the effect of neomycin was only on the secondary infection.

DR. EUGENE F. TRAUB: Is this case simply one of mild pemphigus vulgaris which now has localized lesions occurring on the scalp, or what is the prognosis? I do not quite understand how a patient could have had generalized pemphigus and now have lesions only on the scalp.

DR. LESLIE P. BARKER: It is amazing that one sees a case of pemphigus apparently arrested. The scar on the scalp is probably due to secondary infection or some cause other than pemphigus. Whenever a case of pemphigus remains clear without therapy, one is led to suspect the original diagnosis.

DR. EUGENE F. TRAUB: He presented definite bullous lesions on the hair margin. I wonder whether the diagnosis is possibly Hailey-Hailey pemphigus or, in view of the scarring, bullous lupus erythematosus, or whether he had a great deal of secondary infection. Now the disease looks almost like folliculitis decalvans or alopecia cicatrisata with bullous lesions still present.

DR. WILBERT SACHS: I would rather call it pseudopelade than alopecia cicatrisata. It is possible that the man also had pemphigus in this area.

Boeck's Sarcoid. Presented by DR. LESLIE P. BARKER.

T. S., a Negro woman, is presented from St. Luke's Hospital complaining of subcutaneous nodules of five months' duration. She has had constant fatigue with periodic attacks of nausea for the past two years. Erythrocyte sedimentation rates during 1950 and 1951 were 6, 5, 7 and 4 mm. in one hour. Roentgenograms of her chest showed a normal condition; the blood chemical constituents, including serum protein, urea, uric acid and sugar, were all normal. Her basal metabolic rate was —18%, and she had moderate secondary anemia. The urine was normal. The fatigue was thought to be due to secondary anemia and to low thyroid function. She was given thyroid, iron, and vitamins by mouth. Her fatigue improved only slightly, but the nausea disappeared. Her basal metabolic rate rose to —3%, and periodic determinations have been about the same since.

Five months ago the patient noticed several painless nodules in each antecubital fossa, and at about the same time there appeared a subcutaneous, slightly tender nodule on the left forearm and two on the right foot. These lesions have slowly increased in size. She has had no cough or night sweats.

In 1928 the patient had a stillborn child and in 1937 received antisyphilitic treatment at the Board of Health. Her blood serologic tests have been negative since that time. Examination in 1937 showed a normal spinal fluid.

When first seen at St. Luke's Hospital, on Feb. 12, 1952, the patient showed a cluster of several split-pea to pea-sized cutaneous, reddish-brown, firm nodules in each antecubital fossa. She attributed these lesions to needle punctures, where she had received intravenous injections. She also had a firm subcutaneous nodule, about 3 cm. in diameter, on the external surface of the left forearm and a similar lesion on the right side. On the right heel at about the area of pressure of the top of her shoe there was a pea-sized, firm cutaneous nodule.

The blood count was normal; the blood Wassermann reaction was negative, and the erythrocyte sedimentation rate was 12 mm. in one hour. The basal metabolic rate was —3%. Roentgenograms of the chest showed increase in the hilar areas bilaterally, probably representing hilar adenopathy. Roentgenograms of the hands showed no bone pathology. Biopsy of one of the subcutaneous lesions of the left antecubital fossa showed a histologic structure compatible with sarcoid.

DISCUSSION

DR. EUGENE F. TRAUB: The case is a difficult one to discuss in view of the positive tuberculin reaction, unless the condition is passing over into the phase of tuberculosis, if one accepts that theory. It might be well to x-ray the lungs.

DR. FRANK E. CORMIA: Occasionally corticotropin will make the reaction to tuberculin negative.

DR. WILBERT SACHS: Dr. Barker showed me the slide, and I thought the lesion looked like Boeck's sarcoid. In view of the fact that the tuberculin reaction is positive, I wonder whether in this case tuberculosis may not develop, with all types of reactions.

DR. GEORGE M. LEWIS: Corticotropin or cortisone should not be used in treating this patient because of the danger of activating latent tuberculosis. A positive tuberculin reaction is not unknown in cases of sarcoid; I have seen at least half a dozen such cases, and there are many such reports. This in itself should not exclude a diagnosis of Boeck's sarcoid.

DR. LESLIE P. BARKER: I agree that a patient with a positive tuberculin reaction should not be given cortisone. I have seen several cases of typical Boeck sarcoid in which the tuberculin reaction was positive, but the lesion was usually a little lower than this one. About a year ago some patients with sarcoidosis (Siltzbach, L. E.; Posner, A., and Medine, M. M.: J. A. M. A. **147**:927-929, 1951. Lovelock, F. J., and Stone, D. J.: ibid. **147**:930-932, 1951. Small, M. J.: ibid. **147**:932-937, 1951.) had had prolonged sarcoidosis and showed remarkable improvement after receiving cortisone for six weeks. In one patient tuberculosis of the lungs and bladder developed. One had a positive tuberculin reaction before the cortisone was given. I should certainly not give this patient cortisone until much more study had been carried out.

Multiple Hemorrhagic Hemangioma (Kaposi's Sarcoma). Presented by DR. ANTHONY C. CIPOLLARO.

W. W., a man aged 64, first attended the dermatology clinic of the New York Polyclinic Medical School and Hospital because of lesions on his left foot and leg. He states that the lesions first appeared about three months before and that there has been slow progression in their size. He has never had any previous affliction of the skin other than a wart, which was removed four years ago from the dorsum of his left hand and which he was told was not malignant. The patient has had diabetes mellitus for the past 12 years, which he has controlled by diet. His general health otherwise appears to be good. He has had a prostatectomy and an appendectomy.

On the plantar surface of the left foot is a marble-sized, bluish-reddish mass, situated just posterior to the transverse arch. On the sole and the lateral surface are many small, firm,

elevated, bluish nodules about the size of a BB shot. Also on the lateral surface are sharply demarcated, nonelevated bluish areas. Some of the nodules on the dorsum extend up to the ankle. A lesion about the size of a small pea, whose appearance is the same as those on the foot, is seen on the dorsal surface of the left hand.

The patient has just been seen for the first time, and he has not been completely studied. Biopsy will be performed and the lesions will be treated with irradiation.

NOTE: One month after presentation biopsy confirmed the clinical diagnosis, and the large melanoma-like lesion on the plantar surface involuted completely with superficial x-ray therapy.

DISCUSSION

DR. EUGENE F. TRAUB: I think the presenter's diagnosis is the acceptable one, but the lesions in the center of the instep looked like nevocarcinoma with skin metastases. If it were not for the other lesions, especially the little lesion on the hand, I should still believe that Kaposi's sarcoma was the diagnosis. With all those little nodules on the foot that appeared to be inflammatory, the diagnosis of Kaposis sarcoma is undoubtedly correct.

DR. PAUL E. BECHET, Elizabeth, N. J.: I agree with the diagnosis.

DR. WILBERT SACHS: If the biopsy shows Kaposi's sarcoma, I should accept the clinical diagnosis. I would say that the prognosis is not good and that life expectancy may be comparatively short because of the widespread lesions. Lesions of Kaposi's sarcoma might very easily develop internally.

DR. FRANK E. CORMIA: Has anyone ever observed melanocarcinoma occurring during the course of Kaposi's sarcoma?

DR. GEORGE M. LEWIS: The manner of spread is not quite that of melanoma, and I believe this disease may be ruled out clinically. I think it is Kaposi's sarcoma, but it certainly is an early stage.

Lymphoblastoma (Mycosis Fungoides?). Presented by DR. FRANK E. CORMIA.

C. H., a Russian-American man aged 63, has been bothered with swellings of the legs, feet, and hands for nine months. At about the same time a flat, erythematous eruption has been noted. This eruption was originally scattered over trunk and extremities. Lesions occurred singly or in groups and lasted for a day or two, or as long as three weeks. They were intensely pruritic and itched easily when traumatized. More recently they have become more infiltrated and somewhat larger. Local treatment has been without value. Six months ago he was given large doses of tripelennamine (pyribenzamine®) hydrochloride, without effect. More recently a trial of chlorprophenpyridamine (chlortrimeton®) maleate was similarly ineffectual, and he was given cortisone, 100 mg. daily, for two weeks. This alleviated the itching, but the lesions were not altered.

Scattered over the trunk and extremities, more plentifully on their proximal portions, are rather large, infiltrated plaques; these are darkly erythematous, slightly infiltrated, and lichenified, and in certain areas they resemble lesions of chronic urticaria. In addition, there is a considerable amount of spotty hyperpigmentation on the extremities; this, the patient states, has been present for the past year. In addition, there is a considerable amount of edema of the distal extremities. There was moderate hepatosplenomegaly.

Examination of the bone marrow revealed essentially a normal condition, as did an electrocardiogram and a gastrointestinal series. A roentgenogram of the chest revealed some evidence of old tuberculosis at the left first anterior interspace. Blood counts, urinalyses, and blood chemistry examinations gave normal results. The basal metabolic rate was + 11 %.

Biopsy of skin and muscle showed a normal histologic pattern of the muscle fibers, and no infiltrative or degenerative processes were noted. Throughout the thickened, edematous, collagenous layer of the skin were numerous collections of large lymphocytes, arranged mainly around the blood vessels, and also in the subcutaneous fat. Among the lymphocytes were occasional eosinophilic polymorphonuclear leucocytes and large mononuclear fibrotic cells. The epidermis was thin. There were considerable pigment in the epidermis and a moderate amount of keratin on the surface. No definite diagnosis could be made, but the infiltration in the dermis was considered compatible with a malignant lymphoblastoma.

DISCUSSION

DR. LESLIE P. BARKER: If this case proves to be mycosis fungoides (lymphosarcoma), it merely demonstrates the multiplicity of cutaneous lesions that may appear in the early stages of this disease. It is most unusual for a tumor to develop from the urticarial stage.

DR. PAUL E. BECHET, Elizabeth, N. J.: Some lesions have a livid hue and a circinate shape with definite infiltration. I would agree, therefore, with the diagnosis of mycosis fungoides.

DR. WILBERT SACHS: I am under the impression that the condition is not only clinically but microscopically mycosis fungoides of the neurodermatitic type. The fact that the process goes deep in the cutis (on cursory examination) would indicate that. Under the low-power lens a great number of large cells can be seen.

DR. GEORGE M. LEWIS: I agree with the diagnosis of mycosis fungoides.

DR. EUGENE F. TRAUB: If there is such an entity as early mycosis fungoides, this is it in classic form. One could see the infiltrated plaques better when the patient walked in than one can on close inspection. In addition, he presents horseshoe-shaped lesions. In a case such as this, if itching is not intolerable I would either withhold x-ray treatments entirely or give a minimal superficial dose to control symptoms, for many of these lesions come and go spontaneously without treatment.

DR. FRANK E. CORMIA: I am in agreement that the diagnosis is lymphoblastoma, but I am not entirely sure that death will be from mycosis fungoides. There is a pleomorphic appearance in biopsy studies, and clinically at least the picture conforms to that of classic mycosis fungoides. However, the man has decided enlargement of his liver and spleen and unexplained edema. While we may occasionally see involvement of the internal organs in mycosis fungoides, it is the exception, and not the rule.

Case for Diagnosis: Papulonecrotic Tuberculid? Dermatitis Factitia? Presented by DR. MAURICE J. COSTELLO.

F. S., a man aged 26, obese (290 lb. [131.5 kg.]), jumped from an elevation on March 3, 1947, fracturing his left tibia. He was hospitalized for 14 months, and during this time an ulcerated area appeared, irregular in shape, approximately 1 in. (2.5 cm.) in diameter, and surrounded by a cyanotic, erythematosquamous, indurated area, measuring 6 by 7 in. (15.3 by 17.8 cm.) in diameter. He claims that prior to this time he had a number of discrete, lentil-sized lesions on the anterior surface of both legs. These appeared while he was with the Army in the Philippine Islands, and he thought they were caused by "jungle rot." The patient admits picking these lesions, but he states that a small papule is always present before he picks or scratches the area.

DISCUSSION

DR. FRANK E. CORMIA: The patient is grossly overweight; for some time edema of the extremities has developed after he stands on his feet, and I believe a stasis factor is present. I am not convinced that the lesion is a tuberculid, although biopsy should be helpful. I had the impression it was pyoderma. Cultures should be made, as well as a biopsy. If the latter does not substantiate the diagnosis of tuberculid, treatment should be given for pyoderma.

DR. C. T. CHIARAMONTE (by invitation), Brooklyn: The lesions are compatible with pyoderma.

DR. WILBERT SACHS: I don't know how this type of pyoderma can be differentiated from papulonecrotic tuberculid. The two lesions may show the same picture histologically.

DR. PAUL E. BECHET, Elizabeth, N. J.: Dermatitis factitia is a remote possibility, but the clinical evidence is in favor of papulonecrotic tuberculid.

DR. LESLIE P. BARKER: From what the patient presents today, it would be impossible to make a diagnosis of papulonecrotic tuberculid. He does, however, have considerable secondary infection in some of the lesions. He has a glandular imbalance and should be studied from that standpoint. I should treat the cutaneous lesions as pyoderma.

DR. GEORGE M. LEWIS: A culture might reveal something unusual, like a diphtheroid. The lesion doesn't look like the usual kind of pyoderma, and the probability of tuberculid is remote.

DR. EUGENE F. TRAUB: There are probably many types of papulonecrotic tuberculid that I am not familiar with, but I remember that in the old days papulonecrotic tuberculids, regardless of size, were conical papules with definite necrotic centers. These lesions appear to be flat—no induration at all—with a depression such as one sees in pyoderma. Therefore, I believe pyoderma is the more likely diagnosis, and I think that if it is difficult to differentiate such a process microscopically, one of the newer antibiotics or a combination used locally might give a better therapeutic diagnosis than anything else.

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Necrobiosis Lipoidica Diabeticorum. Presented by DR. BERTRAM SHAFFER and (by invitation) DR. M. GRAY.

S. K., a white woman aged 57, obese, flushed, with apparent dulling of intellectual faculties, presents on the right leg, just above the calf, a circular lesion about 1 in. (2.5 cm.) in diameter, showing a firm, depressed center with telangiectases and a slightly raised, reddish-yellow border. On the medial aspect of the lower part of the leg there is a similar, smaller lesion with an ulcerated center.

The patient has had hypertension for over 10 years and diabetes for at least 5 years. She takes 20 to 26 units of insulin daily but has had no adequate medical control for several years. The cutaneous lesions appeared about two years ago.

A complete blood cell count revealed nothing remarkable. The fasting blood sugar was 235 mg. and the blood urea nitrogen 14.1 mg., per 100 cc.

The report on the biopsy specimen is as follows:

"The epidermis was normal except for a few small foci of intracellular and intercellular edema. In the midcorium and the deep corium were several foci of early necrobiosis, in which there was loss of nuclei but retention of normal structure of the collagen. These areas were somewhat paler than the adjacent collagen. The surrounding tissue was the site of a moderate diffuse infiltrate, consisting principally of lymphocytes and fibroblasts. The small blood vessels were slightly to moderately thickened, with narrowing of their lumina in several instances."

DISCUSSION

DR. HERMAN BEERMAN: My first thought when I saw the sections was of granuloma annulare. It was only after reading the history, I confess, that I thought of necrobiosis lipoidica diabeticorum, and then I saw certain features which were more compatible with that diagnosis, namely, giant cells and blood vessel changes.

DR. H. H. PERLMAN: If there was ever a typical microscopic picture of necrobiosis, it is shown in this case. The blood vessels are being choked out, filled up; they look like blown-up balloon tires, which I think is a very definite sign of necrobiosis lipoidica diabeticorum.

DR. FRED D. WEIDMAN: For a long time when I saw sizable areas of necrosis in a section, there were only two conditions which I thought of; granuloma annulare and necrobiosis lipoidica diabeticorum. However, recently I have encountered other cases which were presented at the Academy meeting last year, in which rheumatoid arthritis nodules have shown a histologic picture almost identical with that of granuloma annulare, but with this difference, that there is much (and old) fibrosis and, in some cases, calcification. Of course, in a very old, chronic disease like rheumatoid arthritis, the fibrosis and calcification are not surprising. This is an additional necrobiotic disease on the dermatologic map—not "rheumatic nodules" but the "nodules of rheumatoid arthritis."

DR. HERMAN BEERMAN: Every time I take a section of granuloma annulare, or of necrobiosis lipoidica diabeticorum, or of rheumatoid arthritis to Dr. Horn, the surgical pathologist of University Hospital, he diagnoses it as "rheumatoid arthritis nodule."

Pemphigus Erythematodes (Senear-Usher Syndrome). Presented by DR. COLEMAN JACOBSON (by invitation) and DR. DONALD M. PILLSBURY.

T. B., a white woman aged 49, well developed, with a somewhat rounded face, presents an erythematous, scaling, seborrheiform dermatitis on both cheeks and the left side of the forehead. A few circumscribed, erythematous, seborrhea-like patches are located on the chest. There are circumscribed, hyperpigmented areas on the back, arms, and forearms.

After sunburn at the seashore in July, 1950, a rash developed on her face, arms, trunk, and legs. In December, 1950, she was hospitalized at the New York Skin and Cancer Hospital, where she improved under treatment with cortisone. After cessation of cortisone therapy, the eruption flared anew into a generalized cutaneous distribution. She was treated, with good results, at the Hospital of the University of Pennsylvania from Dec. 23, 1950, until May 3, 1951, with penicillin and corticotropin (ACTH). When, on two occasions, corticotropin was withdrawn for a period of one week, new bullae rapidly appeared. The lesions on the back, arms, and legs have now cleared almost completely, with slight residual hyperpigmentation. She still has an erythematous, scaling, seborrhea-like dermatitis of both cheeks and the left side of the forehead, and a few circumscribed areas on the chest.

A complete blood cell count was essentially within normal limits. The blood sugar varied from 90 to 150 mg. per 100 cc. The findings on urinalysis were generally within normal limits, with an occasional trace of albumin and sugar. Recently, the 17-ketosteroid excretion and eosinophile counts showed no laboratory response to corticotropin.

The report on the biopsy was as follows: "The results of a Tzanck test were compatible with pemphigus and biopsy of a specimen from the same bulla was reported as bullous dermatitis."

DISCUSSION

DR. FREDERICK URBACH (by invitation): This case is one of our longest follow-up cases of corticotropin therapy. The patient has been treated now for almost 15 months with intravenous injections of corticotropin, and whenever this therapy is discontinued or changed to placebo therapy, which we have done on a number of occasions, she breaks out within 72 hours with new bullous lesions. Interestingly enough, from the biochemical point of view, this patient shows no evidence of adrenal stimulation. The drug does not affect the eosinophile count. We gave her saline, without her knowing that it was simply saline, and again in three days she had new lesions. It would appear that patients who have received corticotropin for long periods will lose all evidence of biochemical stimulation of the adrenals; yet the clinical picture is that of remission. One must be careful therefore in evaluating corticotropin from the biochemical evidence and must go by the clinical picture presented by the patient. I may also say that this patient has had no ill effects from this prolonged administration other than a very mild early Cushing syndrome.

DR. BERTRAM SHAFFER: Dr. Pillsbury and I used corticotropin in treating a patient recently with the Sulzberger-Garbe syndrome who was already mildly diabetic, and we found that a slight increase in insulin was required. In other words, it is possible to give such a preparation as corticotropin to patients with mild diabetes, provided it is adequately covered with insulin.

Delayed Angiomatous Zosteriform Nevus. Presented by DR. J. M. SCHILDKRAUT, Trenton, N. J.

M. S., a white girl aged 10 years, presents on the anterior surface of the right side of the chest, extending from the fourth to the seventh rib, a macular, small, blotchy, reddish-brown eruption, which is angiomatous in nature. It is zosteriform, following the line of the ribs, with a few lesions in the right axilla and on the right side of the chest posteriorly. There is no history of any vesicular or other eruption preceding this. It was first noticed in July, 1951, beginning in the right axilla and spreading onto the chest.

DISCUSSION

DR. THOMAS BUTTERWORTH, Reading: I think that time will give us the ultimate diagnosis in this particular case, but I believe that Majocchi's disease (purpura annularis telangiectodes)

should be considered. In this plaque one observes telangiectasis, purpura, and in some areas, I think, atrophy; in two places there is a suggestion of ring formation. I realize that Majocchi's disease occurs on the abdomen, flanks, and lower back. I have never seen it in zosteriform distribution, but I am offering it as an alternative diagnosis.

DR. FRED D. WEIDMAN: I agree that the basis for this nevus is capillaries; it is not a nevus in the sense of the usual papillomatous nevus, nor is it the epidermal type of nevus, in which it is only the epidermis that is congenitally abnormal. There is another dermatosis which can be zosteriform, and that is leiomyoma. We have seen two such cases in this society, in which the involuntary muscle was significantly related to hair follicles. In such a case, of course, the lesion would classify as a hamartomatous expression and fall into the nevus category. I think that this is a capillary nevus only in the broad sense and that, in view of a certain background of organization (organoid), that is, organization to the extent that there is a relationship to some anatomic structure, in this case the intercostal nerves, this lesion may be one more of those hamartomatous kinds of nevus change. There is a connection in the pathogenesis of the zosteriform leiomyomas and this particular lesion.

DR. JOHN M. SIEGEL, Allentown: I would advance the diagnosis of a very early form of atypical poikiloderma vasculare atrophicans. That has the very same picture that Dr. Butterworth describes.

Trichophyton Rubrum Infection. Presented by DR. A. M. KLIGMAN and (by invitation) DR. M. GRAY.

J. G., a white man aged 32, presents a scaling, fissuring eruption of the right palm. Lesions had been present on the right hand and foot for eight or nine years, with partial remissions. The patient was first seen by the presenters on Aug. 16, 1951. At this time there were some maceration of the skin between the fourth and fifth toes of the right foot and scaling of the right palm, together with small, deep-seated vesicles along the lateral aspect of the fingers.

The serologic reactions for syphilis were negative. Scrapings for fungi on Aug. 16, 1951 were also negative. Scrapings taken on Oct. 15, 1951, were positive for fungi.

Despite the negative results in scrapings in the first examination, the patient was treated with "desenex" ointment and powder (a preparation of undecylenic acid and zinc either in an ointment base or talc) to the feet and aluminum acetate solution U. S. P. (Burow's solution) soaks to the hand. This was followed by improvement, but scaling of the hand continued, despite ultraviolet radiation therapy and the application of benzoic and salicylic acid ointment U. S. P. (Whitfield's ointment) and tar preparations. The patient works as an assembler of telephone panels and showed no change when an occlusive glove was worn at work.

DISCUSSION

DR. HERMAN BEERMAN: I think this is a remarkable diagnosis. Has Dr. Kligman any criteria by which we might be able to make it clinically?

DR. ALBERT M. KLIGMAN: This type of lesion is rather characteristic. It is remarkable that it is so often unilateral. Moreover, fungous elements are frequently difficult to detect. In this particular case, a very considerable quantity of scrapings removed from various parts of the hand revealed small hyphal segments in the smear stained with periodic acid and fuchsin. It seems remarkable that hyperkeratosis as diffuse as this can be due to so few fungous elements. Perhaps there is some more fundamental disturbance, on which the fungous infection is simply superimposed. These lesions are extraordinarily difficult to treat and generally persist for an indefinitely long period.

DR. REUBEN FRIEDMAN: We had a patient in the office about 10 days ago, a telephone worker who used his right hand in holding cables and making repairs. He presented a diffuse scaling dermatitis identical with this patient's. Scrapings showed many mycelia under the microscope.

DR. REUBEN SCHWARTZ: Should we change our concept of the primary fungous infections of the hand? I thought they were rare.

DR. M. H. SAMITZ: About a year ago Albom and I (Allergic and Nonallergic Causes of Eczematous Hand Eruptions, *Ann. Allergy* **9**:336, 1951) reported nearly 400 cases of eruptions

of the hand, in 14 of which we found primary fungous infections. Since then we have had 54 cases identical with these, including several instances of bilateral involvement. I think that this condition is commoner than is reported. We found Trichophyton rubrum infection of the hand in an incidence as high as food allergy of the hand. Scrapings from between the thumb and the index finger will frequently demonstrate the fungus. I have found anthralin to be the best drug, beginning with 0.25% and increasing to 1%.

DR. HERMAN KLINE, Atlantic City, N. J.: Have any of these cases been found in women? I have seen two or three, but all of men.

DR. JOHN H. COX (by invitation): I, too, was taught that primary fungous disease of the hand is extremely rare, but I am certain I have seen 15 or 20 cases during a recent two-year stay in Hawaii; and since I have been in Philadelphia I am sure I have seen 12 cases.

DR. FRED D. WEIDMAN: This is a surprise to me, too. Why have so many of the discussers testified to the frequency of these cases? There is a ready answer, namely, that the styles do change in mycology. The predominating species, for example, of a fungus in a given geographical locality will change within a single generation. The older mycologists recall the time when Epidermophyton floccosum was the predominant species for ringworm of the groin, and the predominant species in ringworm of the toes was Trichophyton gypseum. Now the tide has turned, and we are seeing more T. rubrum infections. This is running true to form; processes in mycology change in a way that is more extreme than in other classes of disease.

Inflammatory Histiocytoma: Nevoxanthoendothelioma. Presented by DR. MEYER L. NIEDELMAN.

P. L., a Negro boy aged 3 months, presents a group of tumors on the right side of the scalp extending from the hair line backward and to each side about 1½ in. (3.8 cm). They appeared shortly after birth and have increased in number and size. They vary in size from several millimeters to 1 cm. and are definitely circumscribed, the color varying from pearly-gray to distinct yellow. They are hard and noninflammatory, with no tendency to undergo involution. Two lesions have a denuded surface. The entire area is devoid of hair. The intervening skin is smooth and glossy.

Dr. Fred Weidman's report on the biopsy specimen follows: "The epidermis was slightly acanthotic, and, for an infant, was slightly hyperkeratotic. In the corium, the normal collagenous patterning had been replaced largely by bundles of spindle-shaped cells, which were surrounding capillaries, together with great numbers of individual cells of indeterminate nature. Such cells were evidently not epithelial, but, after that, it was a question whether they were histiocytes or degenerated epithelioid cells. In any event, the cytoplasm of the cells had become granular, violaceous, and fused with that of neighboring cells, so that the outline of the cell was no longer distinguishable. There were no foam cells, capillary hyperplasia, or multinucleated cells of nevoxanthoendothelioma.

"*Summary and Comment.*—The feature is a hyperplasia of histiocytes, with such vast degeneration of the cytoplasm that their identity is arrived at largely by exclusion. The degeneration has proceeded so far that it has amounted to a fatty degeneration, probably, affecting the cytoplasm. This conclusion is justified by the yellow color observed clinically and by the extreme grade of degenerative processes.

"In making the diagnosis, I am not implying that the processes are neoplastic. In fact, the acanthosis and the edema point to an inflammatory basis, but I must admit that I cannot designate any particular dermatologic entity which would fit the histologic situation. The pathologic diagnosis is inflammatory histiocytoma."

DISCUSSION

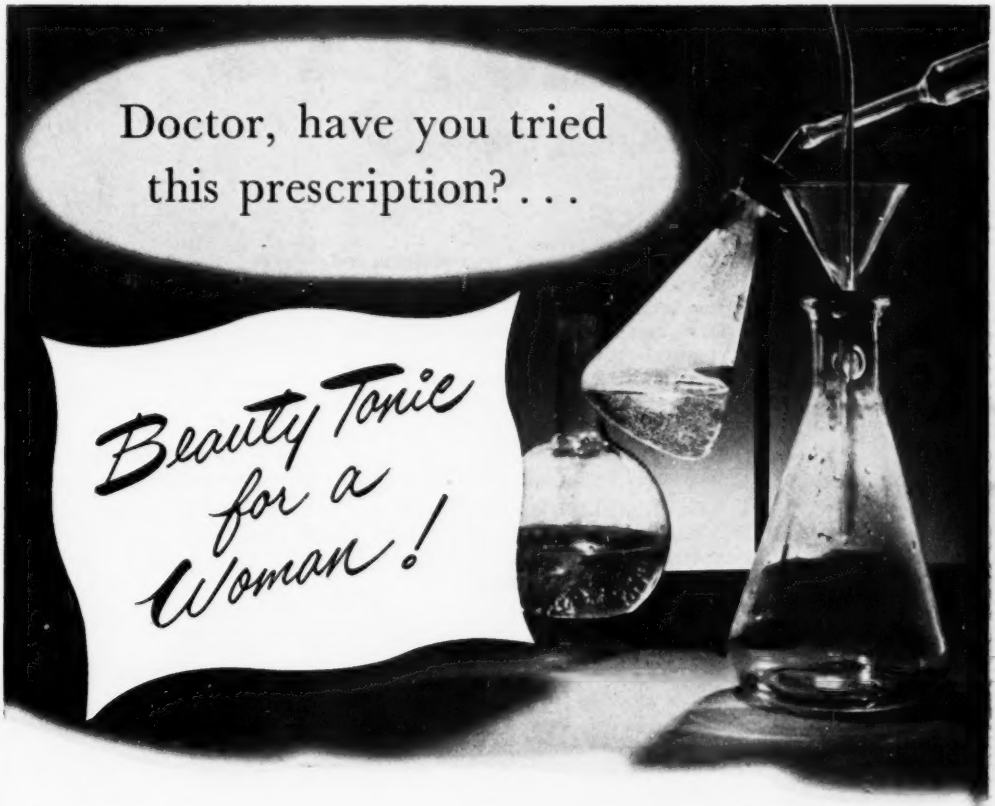
DR. FRED D. WEIDMAN: I saw the section, and so did Dr. Beerman. Clinically, it looks like a nevoxanthoendothelioma, but histologically there is nothing to recommend that diagnosis. There are not any true foam cells or any hyperplastic capillaries. McDonough is not a dermatologist; he is a general pathologist. He gave the name on the surmise that the hyperplastic capillaries were the origin of the tremendous proliferation that he saw. Therefore, without capillary proliferation there cannot be any nevoxanthoendothelioma. The term "nevoxantho-

endothelioma" does not connote a special kind of nevus cell but is used to designate a congenital anomaly. From what I have seen of these lesions, the processes are not those of a neoplasm. They make me think much more of some abnormality of the leucocytic system, and the foam cells in the case are probably those of a lipid dystrophy rather than an abnormality of neoplastic cells; these cells are reticuloendothelial cells in the final analysis. In view of so many cells that do not look at all like the cells of endothelium or capillaries, I contend that at present this lesion does not have the histology of nevoxanthoendothelioma. I can understand how in frozen sections fat would appear, because the large, swollen cells are highly degenerate. The closest approach I could make was that these cells were lipid histiocytes and that this lesion is more like a histiocytoma than a nevoxanthoendothelioma.

DR. HERMAN BEERMAN: I am very much interested in nevoxanthoendothelioma from the clinical standpoint. I think that this case represents a cross between Hand-Schüller-Christian disease (lipid histiocytosis of cholesterol type), or some related entity, and histiocytoma, and, therefore Dr. Niedelman should have a roentgenogram of the skull to see if there are any bony changes. Dr. Blank reported one such case in which there was ocular involvement. Essentially, I think that Dr. Weidman and I are not in disagreement although I first thought of nevoxanthoendothelioma, rather than histiocytoma. This also brings to attention Dr. Lever's contention that histiocytoma and nevoxanthoendothelioma are the same thing. Dr. Tannhauser has the same idea, namely, that nevoxanthoendothelioma and Hand-Schüller-Christian disease are one and the same, but he never had any real reason to think so. He had no occasion to prove it. We feel that our case lends some evidence to that side.

DR. MEYER L. NIEDELMAN: The differential diagnosis of sclerosing hemangioma, histiocytoma, and nevoxanthoendothelioma usually is made on histologic grounds. That is, in the first condition a great deal of fibrosis is present; in the second there are numerous histiocytes with newly formed capillaries and a few large foreign body giant cells; in the last, in addition to the histiocytes, one sees many typical foam cells, giant cells, and foreign body giant cells. Although clinically these lesions resemble the group that McDonough described as nevoxanthoendothelioma, one must call the lesion in this case a histiocytoma on histologic grounds. Our reason for showing this infant tonight is to inquire as to therapy. I believe that nothing should be done, as I have seen these lesions disappear spontaneously in other patients. However, the parents are insisting on surgery. I believe that plastic surgery may be helpful. I do not believe that the hair will regrow. Has anyone any suggestions? Roentgenograms of the skull and long bones, the eyegrounds, and blood cholesterol and lipid levels were normal. This would rule out the diagnosis of Hand-Schüller-Christian disease at present.

DR. JOHN M. SIEGEL, Allentown: I have seen a case of nevoxanthoendothelioma in a child who had a large number of these tumors all over his body. By the time he reached the age of 6 years the lesions had undergone spontaneous involution, leaving many pock-like scars scattered over his entire body.



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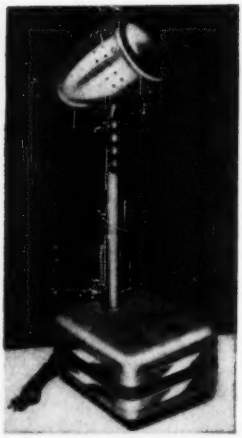

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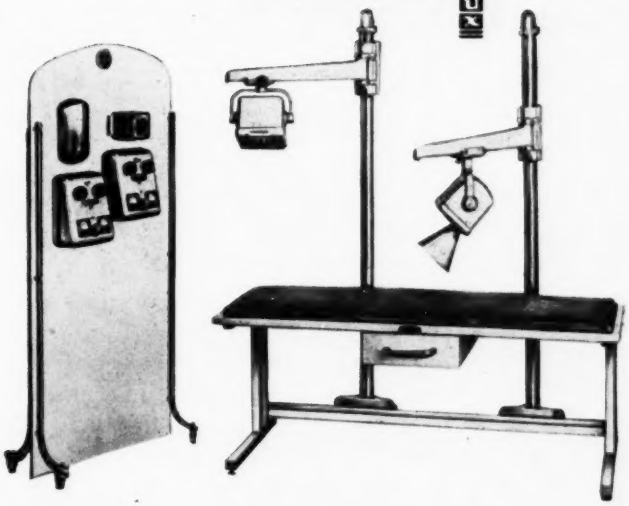

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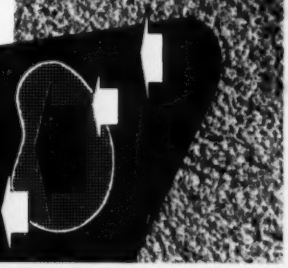

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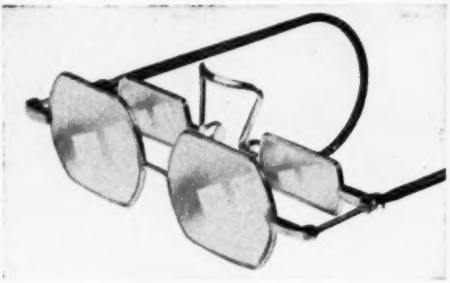

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The Exakta, with instantly interchangeable lenses, is widely used by dermatologists for every type of medical and research photography. Its unique through-the-lens viewing system assures absolutely correct "on-the-subject" photographs, in extreme close-ups or through the microscope. In addition, this world famous medical camera is regularly used for preliminary, developmental, and end result pictures of patients; for copying X-Rays; and for making color transparencies—a necessity for recording and lecturing.

With f2.8 Zeiss Tessar "T" Coated Lens with Pre-Set Diaphragm	**$269.50**
Penta-Prism Eye Level Reflex Viewfinder	**50.00**
Extension Tube Set	**24.00**
Microscope Adapter	**29.50**

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Exclusive Sales and Service Organization in the U.S.A. for Ihagee Camera Works, Germany

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Waterproof, CLOTH-LINED RUBBER GLOVES

Excellent protection where *dishwashing, laundering, cleaning, dusting, painting, gardening, etc.,* may cause dermatitis or intensify skin disorders.

Soft *cotton-lining* absorbs perspiration, prevents contact with rubber, makes gloves slip on and off easily.

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Myristic and lauric acids saponified with polyamine alkali to a low pH(8), with neutralized polycarboxylic acid, enriched with lanolin esters, given extraordinary solvency with propylene glycol.

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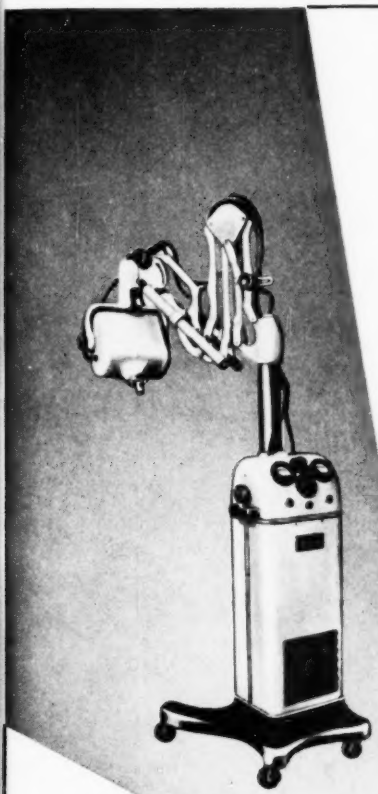

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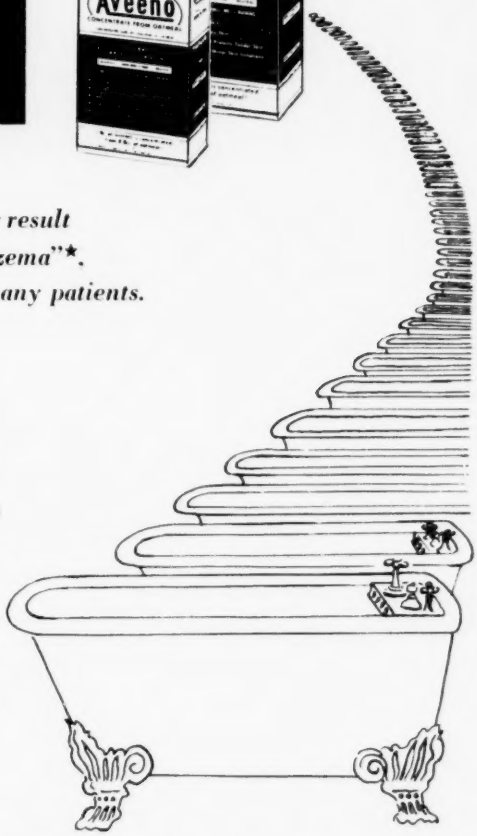
★ Sulzberger, M. B. and Baer, R. L.: Medical Uses of Soap, J. B. Lippincott Company, Philadelphia, 1946



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